بسمـــ الله الرحمن الرحيمـــ

فريق عمل كل الطب

يُقدمـــ

سلسلة كتب د/أحمد موافي

In Capsule Series

تم الرفع بواسطة فريق عمل كل الطب

ALLTEB MEDICAL TEAM

مجمع ومنقول من أكثر من مصدر

جزى الله خيرا كل من ساهم في هذا العمل

لا تنسونا من صالح دعائكمـــ،،

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In Capsule Series

Internal Medicine

NEUROLOGY

First edition

by

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and

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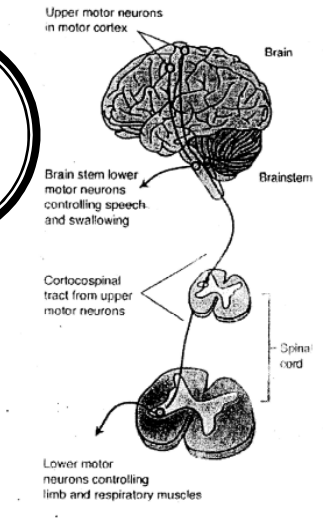
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Hemiplegia

Definition:

Paralysis or paresis of one upper & one lower limb of **the same side** of the body due to **UMNL** above **C5** .

UMNL: any lesion above AHC.
LMNL: any lesion in AHC down to the muscle.



features	UMNL	LMNL
1.Paralysis	**On the contralateral side** if the lesion is above the pyramidal decussation **On the same side** if the lesion below the pyramidal decussation	On the same side of the lesion
2.Wasting of muscles	**No** wasting (disuse atrophy)	**Wasting** of the muscles early
3.Reflexes	**Hyper**reflexia	**Hypo**reflexia
4.Muscle tone	**Hyper**tonia	**Hypo**tonia
5.Babiniski sign (planter response)	**+ve**	**-Ve planter response**

Aetiology:

1-vascular : the commonest cause of hemiplegia.

a-Thrombosis : cerebral atherosclerosis

b-Embolism : either from heart : MS,MI,AF,IE

or vessels: carotid vessels

c-Haemorrhage:

Haemorrhagic blood diseases: purpura

Hypertension

Aneurism

Angiomatous malformation

Trauma

Tumor

	Thrombosis	Embolism	Haemorrhage
1- Age	Old	Young	Old
2- Onset	Gradual	Sudden(dramatic)	Sudden
3- Prodroma	Yes	No	No
4- Vomiting	Not common	Not common	Common
5- Consciousness	Usu ally normal	Usually normal	Deep coma
6- Convulsion	Rare	Rare	Common
7- Heart	IHD	MS,MI,AF,IE	Lt.ventricular hypertrophy,
8- Blood pressure	May be high	Normal	HTN
9- CSF	Clear	Clear	Usually high
10- CT, MRI	Hypodense	Hypodense	Bloody Hyperdense

2-Congenital : cerebral palsy

3- Trauma

4-Inflamatory : meningitis , encephalitis

5-Neoplastic

6-Degenirative : multiple sclerosis

Stages of Hemiplegia

1) Stage of shock (flaccid paralysis)

- Occur only in cases with sudden onset as embolism – trauma and absent in gradually developing cases
- Duration varies from seconds to months
- The more the prolongation of this case, the worse the prognosis
- In this stage, the paralyzed muscles show the criteria of LMNL Except Babiniski sign is **+ve**

2) Stage of spastic paralysis

- It is the stage of established Hemiplegia
- The paralyzed muscles show the criteria of UMNL

N.B

It affects the Distal more than Proximal muscles

So, skilled movements of fingers & toes are More affected

U.L → Extensors are weaker than Flexsors

L.L → flexors are weaker than Extensors

Levels of Hemiplegia

❶ Cortical hemiplegia

1) Starts usually as contralateral Monoplegia not hemiplegia
   It is due to wide distributions of Betz cells in area 4
2) History of Convulsions before the onset of paralysis (irritation in area 4)
3) **Coma** if lesion is extensive
4) Other cortical manifestations
   - Contralateral cortical sensory loss
   - Aphasia
   - Homonymous hemianopia

❷ Subcorical hemiplegia

- ✔ The same as cortical **but** the paralysis is **more extensive**
- ✔ **Coma and convulsions** are less extensive

❸ Capsular hemiplegia

1) ***Complete hemiplegia*** from the start on the opposite side
2) ***Hemianasthesia*** in the opposite side of lesion (same side of the paralysis)
3) ***Homonymous hemianopia***
4) No **C**oma, No **C**onvulsions, No other **C**ortical manifestations
5) Paralysis of the muscles of the lower part of the face **on the opposite side of the lesion** (the Same side of hemiplegia)
6) Hypoglossal paralysis on the same side of the lesion (Deviation of the tongue to the side of paralysis)

N.B All motor muscles receive bilateral pyramidal supply except the lower half of 7^{th}&12^{th}.

❹ Brain stem hemiplegia(Crossed hemiplegia)

1) Hemiplegia is on the opposite side of the lesion
2) Motor cranial nerve affection of the same side of the lesion due to affection of the cranial nuclei in the Brain stem(LMNL)

N.B The cranial nerve affection differs according to the site of the lesion in the brainstem

➲ Mid brain lesion

Ipsilateral **3^{rd}** & **4^{th}** cranial nerve paralysis + contralateral hemiplegia

➲ Pontine lesion

Ipsilateral **5^{th} ,6^{th} ,7^{th}** cranial nerve paralysis + contralateral hemiplegia

➲ Medullary lesion

Ipsilateral **9^{th}, 10^{th}, 11^{th}, 12^{th}** cranial nerve paralysis + contralateral hemiplegia

❺ Spinal hemiplegia

1) Hemiplegia on the same side of lesion
2) Contralateral loss of pain & temperature sensation
3) Touch sensation is often clinically normal

Complications due to prolonged bed rest

1- Psychosis
2-Bed sores
3-DVT
4- Wasting of the muscles (Diuse atrophy)
5- Retention of urine (Not organic but psychic)
6-Constipation
7- Aspiration pneumonia

Investigasions

1- CT
2- MRI
3- Echo: for cardiac causes of embolism.
4- Doppler on the carotid vessels.

Leading Causes of Death

Causes of death after stroke is not related primarily to the stroke itself.

- Pnumonia.
- Pulmonary embolism.
- Ischemic heart disease.

N.B

Value of examination of the cranial nerves

- If there is no cranial involvement: the lesion is **spinal hemiplegia.**

- If the only cranial nerve involved is the lower part of 7^{th}&12^{th}: the lesion is usually **capsular hemiplegia.**

- If the cranial nerve is LMNL&affected the opposite side of hemiplegia: the lesion is **brainstem hemiplegia.**

TREATMENT

1-General Care of the comatosed patient 5B

-**B**ed: frequent change in the position to prevent bed sores.
-**B**alanced nutrition : tube feeding.
-**B**ladder : inserting urinary catheter.
-**B**owel : daily enema.
-**B**reath : (care of respiration)
- O2 inhalation
- suction of secretions
- antibiotics to prevent chest infection (pneumonia)

2-Sypatomatic treatement

- Cerebral dehydrating agents: (manitol+lasix)
- Antiemetics
- Antipyritics: (paracetamol)
- Sedative ,tranquilizers
- Muscle relazents for spasticiy

3-Physiotherapy

- Regular passive exercise of the paralyzed limbs.
- Positioning of the paralyzed limbs in the optimal position.

4-Control of blood pressure

- Hypertension may be of a high benefit as it helps to maintain blood flow to ischemic areas.

So , Antihypertensive drugs are only indicated **when** systemic pressure is more than 220mmHg **or** diastolic pressure is more than 140mmHg

5-Specific treatement (treatment of the cause)

A-Thrombolytic Therapy

- Tissue plasminogen activators (TPA) is indicated in a case of thrombosis (0.9mg/kg infused over one hour)

It should be used as soon aspossible (within 3 hours) from the onset of symptoms.

- Remember that time is brain,No...time is a lot of brain!!
- There should be no contraindications such as haemorrhage , hypertension

Few patients,In fact,fulfill these criteria.

B- Anticoagulants

-Indicated only in embolic cases with no other contraindications for there use as haemorrhage

Methods:

- **Heparin** (5000u/ml) infusion for 2 days
- **Oral Anticoagulants** (marevan): begin at the same time with heparin (action start after 2 days)

As follow

1st day	5mg t.d.s
2nd day	5mg twice
then	5mg daily for 2 months

C- AntiPlatelets

-Aspirin is highly recommended for all cases of ischemic stroke.

- Aspirin reduces the recurrence & mortality rate.

D-Cerebral Haemorrhage

- Evacuation of blood surgically provided that it is not deep haemorrhage.
-Antifibrinolytics : Epsilon amino carponic acid.

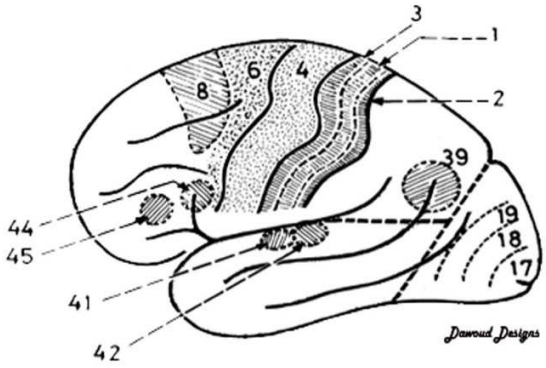
6-neuroprotective *Not Effective*

As; *CerebroLysin* : nerve growth factor

Trental,Nootropil : decrease O2 consumpion & blood viscosity.

Areas of cerebral cortex

Frontal lobe:



- Area 4: infront of the central sulcus.
  Upside down presentation.
- Area 6: posterior to the precentral sulcus.
- Area 8: in the middle frontal gyrus.
- Area 44,45: in the dominant hemisphere.
- Prefrontal area: in the most anterior part.

Aphasia: patient can`t express his ideas in spoken words.
Agraphia: patient can`t express his ideas in written words.

Parietal lobe:

- Area 1,2,3: posterior to the central sulcus.
  Upside down presentation. ⇨

Cortical Sensations

-Midzone temperature.
- Position,Movement.
- Fine touch(tactile localization , tactile discrimination ,streognosis)

- Area 37: infront of the angular sulcus In the dominant hemisphere.
Apraxia: No practice.
- Area 39: Posterior to the angular sulcus in the dominant hemisphere.
Alaxia: can`t read.

Temporal lobe:

- Area 41,42: in the supratemporal gyrus.
- Area 22: adjacent to area 41,42.
- Ancus & hippocampus: smell & memory.

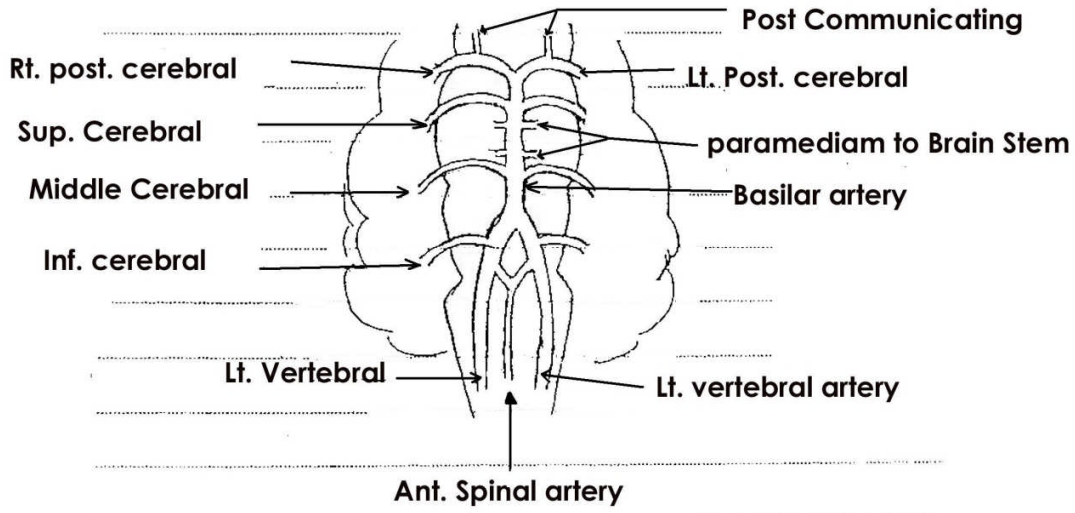
Limbic system ⇨ Uncus & hippocampus
Site: in the medial inferior part of the temporal lobe.
Function: smell & memory.
Leison: - olfactory hallucination
- Loss of memory for the last events

Occipital lobe:

- Area 17: reception of visual images.
- Area 18,19: anterior to area 17.
  Recognition of the images.

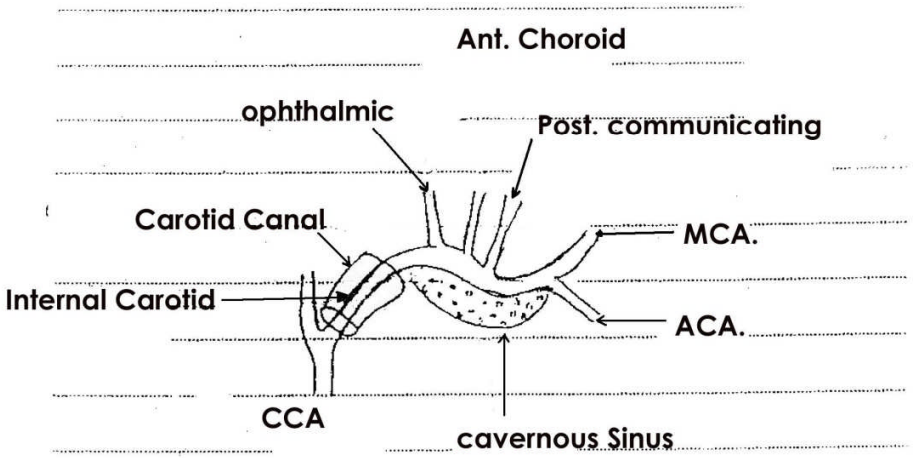
Blood Supply of the Brain

1-Vertebral System



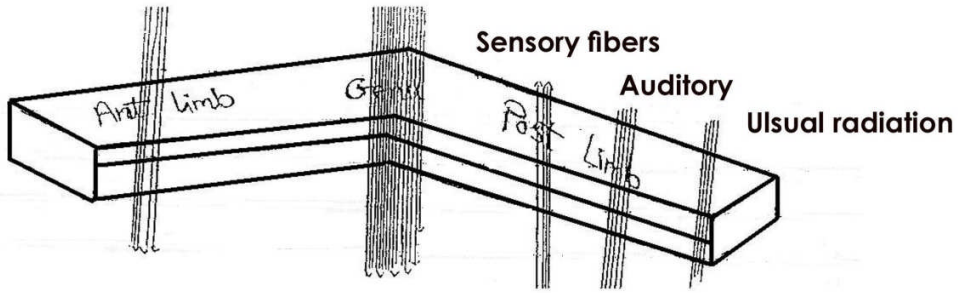
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2- Carotid System



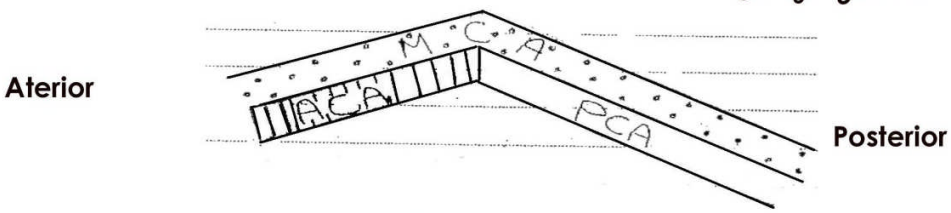
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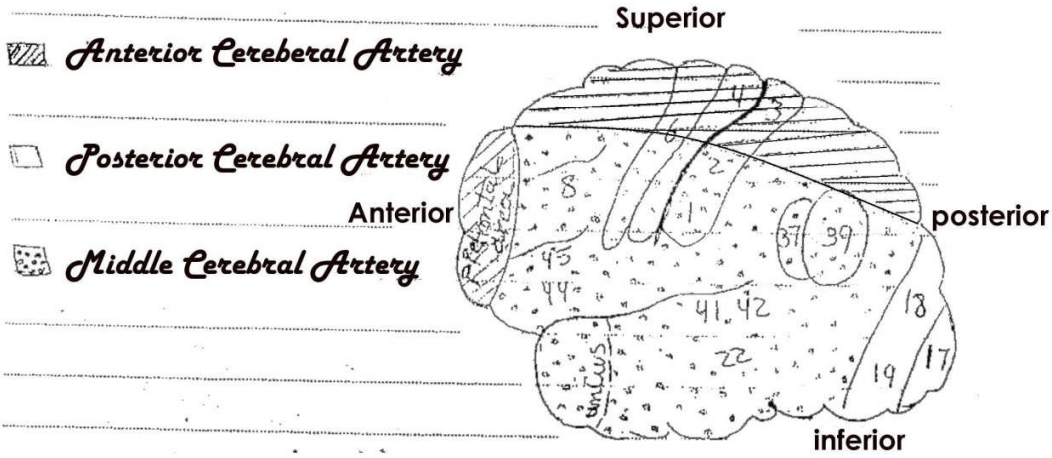


Internal Capsule

Alsayed Dawoud



Blood supply of the Internal Capsule



Cerebral Blood Supply

Vertebrobasilar system occlusion

①anterior spinal artery:

- Quadriplegia if above C5
- Paraplegia if below C5.

②Brainstem branches :

- Brainstem hemiplegia

③Posterior inferior cerebellar artery (PICA):

- Ataxia. وتذكر
- Lateral medullary syndrome

- **Sympathetic**: nasal congestion,Horner`s syndrome,-ve sternospinal reflex. (No pupil dilatation on pressing sternomastoid)
- **Spinothalamic trunk**: loss of pain & temperature from the opposite side.
- **Cranial nerves**: 10th cranial nerve lesion,palatopharyngeolaryngeal manifestations as (nasal tone,nasal regurge, dysphagia, horseness of voice).
- **Vestibular of the 8th cranial nerve** : Vertigo & Vomiting.
- **Spinal nucleus of the trigeminal nerve**: loss of pain & temperature from the same side of the face(crossed hemianaethesia).

4- Anterior inferior cerebellar artery (AICA):

- Ataxia. وتذكر
- Pass in pons ⇨

- **Sympathetic**: as PICA .
- **Spinothalamic trunk**: as PICA .
- **Cranial nerves**: 5th,6th,7th >> see cranial nerves.
- **Cocclear of the 8th cranial nerve** : Deafness.

5- Superior cerebellar artery: (AICA - 5th,6th,7th cranial nerves)

- Ataxia. وتذكر
- sympathetic, spinothalamic, cocclear of 8th cranial nerve.

6-Posterior cerebellar artery:

a- Cortical branch occlusion:

-visual hallucination (area 17)
shoulder,
-H.hemianopia
- Visual agnosia (area 18)

b- Deep branch occlusion:

- Thalamus (Thalamic pain ,Frozen
loss of all sensations)
- Basal ganglia .
(extrapyramidal manifestations)

c- Main system occlusion: a+b

Carotid system occlusion

1- Anterior cerebral artery:

a- cortical branch occlusion:

-- ***Area*** 1,2,3(upper part): monohypothesia of the paralyzed limb.
-- ***Area*** 1,2,3(lowe part): monohypothesia of the paralyzed limb.
-- ***Area*** 4(upper part): contralateral monoplegia.
-- ***Area*** 4(upper part): contralateral monoplegia.
-- ***Area*** 6: extrapyramidal manifestations.
-- ***Area*** 8: disturbed conjugated movement of the eye.
prefrontal Area: change personality and mentality.
-- ***Area*** 44: aphysia.
-- ***Area*** 45: agraphia.
-- ***Area*** 37: apraxia.
-- ***Area*** 39: alaxia.
-- ***Area*** 41,42: auditory hallucination.
-- ***Area*** 22: auditory agnosia.

b- Deep branch occlusion:

Fascioscapular monoplegia

- Proximal more than distal.
- No sensory loss.

N.B: The only case with proximal muscles affected more than distal muscles as the pyramidal tract leison cause the opposite.

2- Middle cerebral artery:

a- cortical branch occlusion:

- Contralateral upper limb monoplegia.
- Contralateral upper limb monohyposthesia.

b- capsular branch occlusion: Capsular hemiplegia

c- The main system occlusion:

How to answer

Vaso-Occlusive Syndrome

Middle Cerebral is the commonest

1) **Anatomy:**
   - *Origin*
   - *Branches and what the supply*
2) **Causes of occlusion:**
   - *Thrombosis and Embolism*→ see Hemiplegia
3) **Clinical picture:**
   - *Cortical*
   - *Capsular*
   - *Main System*
4) **Investigations:** → see Hemiplegia
5) **Complications:** → see Hemiplegia
6) **Treatment:** → see Hemiplegia

Causes of Transient Hemiplegia

- ☑ **T**.I.A (Transient Ischemic Attacks)
- ☑ **E**pilepsy
- ☑ **A**neurysm
- ☑ **H**ysterical
- ☑ **H**ypertensive Encephalopathy
- ☑ **M**igraine
- ☑ **M**ultiple Sclerosis

Stroke

Definition :

- A sudden neurological deficit of vascular origin that persists for longer than 24 hours or leading to death.
- Stroke is the third most common cause of death, after coronary heart disease & cancer.

Types & causes :

I. Ischemic : (Cerebral infarction) *85% of all strokes.*

a) Thrombosis :

- Atherosclerosis : The most common cause
  - ✓ Extracranial : aorta , carotid or vertebral
  - ✓ Intracranial : ACA , MCA , PICA
- Arteritis , Dissection.
- Hematological disorders : Polycythemia, leukemia, sickle cell anemia & hypercoagulability state.

b) Embolism :

- From the heart :
  - ✓ Atrial fibrillation (AF)
  - ✓ Infective endocarditis.
  - ✓ Mitral Stenosis
  - ✓ Myocardial infarction.
  - ✓ Atrial myxoma
  - ✓ Dilated cardiomyopathy
  - ✓ Congenital heart disease.
- From carotid vessels

Risk factors for ischemic stroke

- Risk factors of atherosclerosis : modifiable & non modifiable see cardiology
- Transient ischemic attacks.

II. Hemorrhagic : *accounts for 15% of all strokes*

a. **Intracerebral :** 2 HAT *see hemiplegia*
b. **Subarachnoid** hemorrhage. 2A , 2H , 2T

Clinical feature of ischemic stroke :

1- Focal neurological manifestations : according the occluded artery

*Clinical features of occlusion of the **MCA** or its branches:*

Cortical branch occlusion : Supplies the lower 3/4 of the anterior 3/5 of CC

- Contralateral UL monoplegia with cortical sensory loss.
- Other cortical manifestations : aphasia , agraphia , apraxia (If the left hemisphere is involved)

Capsular branch occlusion : capsular hemiplegia 6 criteria P4

*Clinical features of occlusion of the **ACA** or its branches:*

Cortical branch : supplies upper 1/4 of the anterior 3/5 of the cerebrum

- Contralateral monoplegia in LL with cortical sensory loss
- Mentality & personality changes.

Capsular branch : supplies ventral surface of the anterior limb of the internal capsule.

- Contralateral facio-brachial monoplegia.

Vertebro-basilar system : supplies the posterior 2/5 of the cerebrum , the cerebellum , the brain stem & the spinal cord.

1- anterior spinal artery occlusion:

- Quadriplegia if above C5
- Paraplegia if below C5. (*complete flaccid paraplegia*)

2- Brainstem branches occlusion : Brainstem hemiplegia

3- Posterior inferior cerebellar artery (PICA):

- Ataxia. (……..)
- It is NOT associated with hemiplegia.
- Lateral medullary syndrome (***Wallenberge syndrome***) :

1- Sympathetic: Horner`s syndrome :

- Ptosis - myosis - anhidrosis - enophthalmos - Nasal congestion

2- Spinothalamic tract : loss of pain & temperature from the opposite side.

3- Cranial nerves: 10th cranial nerve lesion → palatopharyngeolaryngeal manifestations as (nasal tone, nasal regurge, dysphagia, horseness of voice).

4- Vestibular of the 8th cranial nerve : Vertigo & Vomiting.

5- Spinal nucleus of the trigeminal nerve: loss of pain & temperature from the same side of the lesion (*crossed hemianaethesia*).

Stages : Flaccid then spastic stage P3

2- Negative in quality :

Loss of function rather than positive (e.g. *Paralysis rather than jerking , blindness rather than visual hallucination*)

3- Rapidly developing : The onset is sudden and acute.

4- Maximum at onset : Over minutes to hours.

5- 80% of stroke patients have at least one vascular risk factor and most are elderly. Stroke is uncommon (but not rare) in young people.

Investigations of ischemic stroke

1- First line investigations :

- CBC & ESR.
- INR , PTT
- Plasma glucose , cholesterol, urea , electrolytes.

- Urinalysis.
- ECG.

2- Cerebral imaging :

i. **CT brain :**
   - To exclude non vascular causes e.g. tumor.
   - To differentiate an ischemic stroke (hypodense blackish lesion) from hemorrhagic stroke (hyperdense whitish lesion)

ii. **MRI :** Usually detects area of evolving infarction within hours, CT is sometimes negative for up to several days after acute infarction.

3- Echocardiography : if cardiogenic embolism is suspected.

4- Duplex carotid ultrasound : When carotid artery occlusion is suspected.

5- Angiography : it plays an important role in the preoperative evaluation of carotid artery disease.

Treatment of ischemic stroke

- **As in hemiplegia : 6 items P7 plus**
- **Carotid endarterectomy for patient with severe carotid stenosis (> 70%)**

1- General care of the comatosed patient : b

2- Symptomatic ttt :

3- Rehabilitation :
   - Physiotherapy
   - Speech therapy.

4- Control of blood pressure.

5- Specific treatment :
   - Thrombolytic therapy (contraindications : in cardiology book p 62)
   - Anticoagulants
   - Anti-platelets

6- Neuroprotective

Prognosis

Complete recovery is uncommon, but the sooner improvement begins, the better the prognosis.

Depends on :

- Site & size of infarction.
- Patient age
- Other comorbidities.
- Impaired consciousness , aphasia , or severe brain stem signs suggest poor prognosis.

Hemorrhagic stroke

Types & Etiology :

1) **Intracerebral hemorrhage :** See above 2H, 2A, 2T
   - Primary intra-cerebral hemorrhage : intracerebral hemorrhage due to chronic hypertension. *"hypertensive ICH"*
   - Chronic hypertension produces fibrinoid necrosis, lipohyalinosis, and medial degeneration, which make the vessels susceptible to rupture.
2) **Subarachnoid hemorrhage :** 2A , 2H , 2T

Clinical picture of intracerebral hemorrhage :

1) ICH occurs characteristically during activity (rarely during sleep).
2) Symptoms typically begin abruptly.
3) The clinical presentation has 2 elements :

a) Increased Intracranial pressure : headache , vomiting , loss of conscious in some cases. Seizures are uncommon at the onset in 7% of cases but are found in 32% of lobar hemorrhages.

b) Specific clinical manifestations : which are related to the site of hematoma.

4) In order of frequency, the most common site of primary cerebral hemorrhage are :

1- Putamen. (*a basal ganglia nucleus*)

2- Lobar hemorrhage : central white matter of frontal, parietal, temporal & occipital.

3- Thalamus.

4- Cerebellum.

5- Pontine hemorrhage.

1- Putamen :

- This is the commonest type.
- Clinical manifestations vary with the size of hemorrhage, but include hemiparesis, headache, and alterations of consciousness.

2- Lobar hemorrhage : Headache is a first and most prominent symptom.

- Frontal hematoma : hemiparesis more in upper limb
- Parietal hematoma : sensorimotor deficit
- Dominant temporal hematoma : fluent aphasia
- Occipital hematoma : homonymous hemianopia.

3- Thalamic hemorrhage : 15%

- hemiparesis or hemiplegia in nearly all cases with severe sensory deficit
- Aphasia may be present with lesion of the dominant side.
- Extension into the subthalamus & high midbrain may cause ocular disturbances (downward deviation of the eyes & irreactive pupil)
- Rupture into the 3rd ventricle may lead to early hydrocephalus.

4- Cerebellar hemorrhage : 10%

- Acute onset of headache , vertigo , vomiting & inability to stand and walk.
- characteristic syndrome of ipsilateral ataxia, LMN facial palsy, and ipsilateral gaze paresis
- Coma & death may occur due to brainstem compression.

5- Pontine hemorrhage : 6%

- Characterized by coma, quadriplegia, absence of light reaction, pinpoint pupil , decerebrate rigidity, respiratory disturbance, tachycardia, hyperthermia.Death usually occurs within few hours.
- Less severe cases arising from hemorrhage confined to one side of the pons are also recognized.

Diagnosis of intracerebral hemorrhage

1) **Clinical feature :** suggested by :
   - Acute hypertension exceeding the patient's chronic hypertensive level.
   - Vomiting at onset.
   - Severe headache.

NB : Although the absence of headache or vomiting does not rule out the diagnosis of ICH, presence of these symptoms is in favor of ICH or SAH as it is reported in less than 10% of occlusive strokes.

   - Focal seizure.
   - Hypertensive changes of the retina.

2) Brain CT : is the procedure of choice to detect the intracerebral hemorrhage (white mass) with surrounding brain edema.
3) MRI.
4) Lumbar puncture : must performed cautiously as it may precipitate or aggravate an impending cerebellar herniation.
5) **Others :** PT , PTT , Platelets count , Liver function tests.

Prognosis

- 1/3 of the patient die in the first 1 to 30 days
- Death occurs due to either :
  1) Marked rise of intracranial pressure ⇨ ↓ brain perfusion.
  2) Compression of vital centre such as hypothalamus , brainstem.

NB : Patients who survive usually show a surprising degree of restoration of function , since in contrast to infarction the hemorrhage pushes the brain tissue rather than destroys it.

Treatment : see hemiplegia

1- Intracerebral : As ischemic infarction but Surgical evacuation of blood instead of thrombolytic & anticoagulant therapy.

2- Subarachnoid hemorrhage : p39 , 40 , 41

Transient ischemic attacks (TIAs)

Definition :

- TIAs are short transient attacks of cerebral ischemia lasting less than 24h
- Most TIAs last only 10 seconds to 15 minutes.
- RIND (Reversible Ischemic Neurological Deficit) : If the attack lasts over 24 h .
- Patients with TIAs have a higher risk of developing a stroke.

Etiology : The same as ischemic infarction

- Cerebral thrombosis.
- Cerebral embolism.

Clinical picture :

- Carotid TIAs : *see before*
- Vertebro-basilar TIAs : *see before*

Investigations : The same as ischemic stroke

Treatment :

I. **Medical :**
   - Antiplatelets : Aspirin , plavix.
   - Anticoagulants : Heparin , Warfarin.
   - Neuroprotective : **Tr**ental , **Tr**ivastal.
   - Treatment of risk factors : e.g. hypertension , DM , hyperlipidemia , smoking.

II. **Surgical :**

Endarterectomy : An incision is made to open the carotid artery , the plaque is removed.

III. **Carotid angioplasty :** Introduction of balloon to dilate the stenotic artery.

Paraplegia

Def:

Paralysis or paresis of both lower limbs due to bilateral upper motor neuron leison(UMNL).

Aetiology:

1- Spinal paraplegia.

A-Focal (paraplegia with a level):

- Compression:
  - Vertebral:
    - ✓ Trauma(fracture)
    - ✓ Tumour (sarcoma)
    - ✓ Infection (TB)
    - ✓ Inflammation (RA)
    - ✓ Deformities (kyphoscoliosis)
    - ✓ Degenerative (spondylosis)
  - Meningeal:
    - ✓ Meningitis
    - ✓ Meningioma
    - ✓ Leukemia
    - ✓ Lymphoma.
  - Cord:
    - ✓ Syringomyelia.
    - ✓ Midline tumour.
- Vascular:
  - Anterior spinal artery occlusion.
- Inflammatory:
  - Transverse myelitis.

B- Disseminated causes. (multifocal)

- Multiple sclerosis.
- Disseminated encephalomyelitis.

C- Systemic causes. (gradual onset,progressive course,symmetrical)

- Heridofamilial: Hereditary ataxia, Hereditary spastic paraplegia.
- Vitamin B12 deficiency.
- Idiopathic: Motor neuron disease.

2-Brainstem paraplegia

- Syringobulbia.
- Midline tumor.

3-Cerebral paraplegia.

Vascular: thrombosis of the unpaired anterior cerebral artery.
Tumour: parasagittal meningioma.
Congenital: Lihle's disease (congenital cerebral diplegia).



Paralysis of both lower limbs due to LMNL is not considered paraplegia but it should be discussed as separate subjects.

e.g: ☑ ***Roots:*** cauda equine.

☑ ***Peripheral nerve:*** peripheral neuropathy.

☑ ***Myoneural junction:*** myasthenia gravis.

☑ ***Muscles:*** myopathy

Flaccid paraplegia:

It is the shock stage of UMNL when the lesion is acute.

e.g: trauma, vascular, t.myelitis

Clinical Picture of Focal Paraplegia

1- At the level.

A-Vertebral manifestations: (if the cause is vertebral)

Localized deformity or tenderness in the spine.

B-Root (Radicular) manifestations:

- Sensory root affection:
  Pain and parasthesia then hyposthesia in the area supplied by the affected root (dermatome).
- Motor root affection:
  Paralysis or paresis of the muscles supplied by the affected root.

2- Below the level.

A-Motor manifestations:

Paraplegia may pass through the following manifestations:

- Shock stage: common in the acute leison e.g:transverse ***myelitis***
- Stage of paraplegia in extension: due to gradual compression of the pyramidal tract , it has UMNL criteria.
- Stage of paraplegia in flexion: due to interruption ***the Extra Pyramidal*** tract as well as the pyramidal tract (+ve mass & Foix reflexes).

✲ **Mass reflex:**
Urination on scratching the medial aspect of the thigh.

✲ **Piere Marie Foix test:**
Done by firm planter flexion leading to sudden flexion of the ***hip and knee***

B-Sensory manifestations:

❖ Extramedullary compression:

1. Sensory level.
2. Below this level, all sensations are lost.
3. Saddle area is the first area to be affected.

N.B

The sacral fibers are the outer ones & the cervical fibers are the inner ones

So,in the extrapyramidal compression ,early sacral affection occurs

But,in the itramedullary compression ,cervical affection is the rule.

- Intramedullary compression:

  Usually affects the lower cervical & upper thoracic fibers.
  (Jacket sensory loss)
  Dissociation of sensation : loss of pain & temprature with normal touch , This is due to interruption of the decussating fibers of spinothalamic tract by midline leison, while touch & deep sensation ascend in the posteior column without decussation.
  Saddle area is the last to be affected.

	Extra-Medullary Compression	Intra-Medullary Compression
Sensory Manifestations	Sensory level Below this level, Loss of all sensations Saddle area is the 1st to be affected	Jacket sensory loss Dissociation of Sensations ... Saddle area is the last to be affected
Myelography	Saddle Shape	Fusiform Shape
C.S.F Pressure	Markedly ↓↓	Lower than the Normal
Queckensted's test	No change in C.S.F pressure	Less rise, less drop
Froin triad	+ve	-ve

C-Sphincteric (Bladder) manifestations:

- Acute: retention with overflow.... See neurogenic bladder
- Gradual: automatic bladder (involuntary,complete,regular)

Investigations:

1-Plain x-ray vertebrae:

The value of x-ray is limited to the vertebral causes e.g: trauma,tumour,pott`s disease.

2-CT scan & MRI: Diagnose the most cases

Taken at the suspected level of the vertebral column, known by the clinical data.

3- Myelography:

Radiography is taken after injecting a certain dye in the subarachnoid space to detect the level of the compression.

- Normally: the dye moves freely.
- In the intramedullary compression: fusiform shape.
- In the extramedullary compression: saddle shape.

4-CSF examination:

- CSF pressure:
  - Normally: 110 – 150 mm.H2O
  - In the intramedullary compression: the pressure is lowe than the normal.
  - In the extramedullary compression: the pressure is markedly diminished even it may be zero in a case of complete block (Dry type).
- Queckenstedt`s test:
  - CSF is drained into the internal jugular vein;
    So,bilateral jugular vein compression leads to rise of CSF pressure
    While release of the compression leads to rapid drop of the pressure to the normal.
  - In the extramedullary compression: there is no change of CSF pressure because there is no free communication ***between C.S.F*** proximal and distal to the block in which the manometer is inserted.
  - In the intramedullary compression: less rise, less drop due to incomplete obstruction.
- CSF analysis: ***Froin Triad*** in the extramedullary compression
  - Yellowish discoloration(xanthochromia): the compression ***leads to*** venous congestion leading to escape of the blodd pigment into CSF causing its color to be yellowish.
  - Spontaneous coagulation: venous congestion leads to rise of the proteins in CSF.
  - Cyto-albuminous dissociation:
    - In the inflammatory condition, both cells & proteins are increased.
    - In this condition, proteins are increased without increase of the cells.

5-Investigations for the cause:

- X-ray chest for T.B .
- Blood picture for leukemia.

How to answer

How to reach the diagnosis in a case of paraplegia??

1 Clinical picture : See page 16.

2 Investigations: See page 17.

3 Determine the level of paraplegia :

- Determine the spinal cord segment: by clinical data e.g: pain & parasthesia around the umblicus means that the leison in the 10th thoracic segment.
- Determine the vertebral level:

Spinal cord segment	*Vertebra*
C1- C4	***the Same***
C5 - C8	*-1*
T1-T6	*-2*
T7-T12	*-3 e.g: T12= T9*
L1,L2	***opposite T 10***
L3,4,5	***opposite T 11***
S1,2	***opposite T 12***
S3,4,5	***opposite L 1***

Treatment

1- General : as hemiplegia.
2- Symptomatic : as hemiplegia.
3- Physiotherapy : as hemiplegia.
4- Specific treatment : (treatment of the cause)

- ✓ Surgical removal of the tumour.
- ✓ Medical treatment of T.B, Leukemia.
- ✓ Cerebral paraplegia (vascular): as hemiplegia.

Qauda Equina

Definition: Collection of lumbosacral roots in lower part of spinal canal.

Conus medullaris : the last 3 sacral segments of spinal cord (S3,4,5)

Epiconus: the above 4 segments of spinal cord (S1,2-L4,5)

Etiology:

1. Congenital → Spina bifida.
2. Trauma→ fr. Dislocation of lumbar vertebrae .
3. Tumor → memingeoma & neurofibroma.
4. Inflammation→ Pott's disease .
5. Degeneration → lumbar spodylosis .

Clinical picture:

1) Motor: *1st convulsions then paralysis or paresis of muscles supplied by the same root;*

# L2	- Flexion of hip	- Iliopsoas
# L3	-Extension of Knee	- Quadriceps
# L4	- Dorsiflexion of ankle	- Ant. Tibial group
# L5	- Dorsiflexion of toes	- Ant. Tibial group& Gltei
# S1	- Planter flexion ankle & toes	- Calf &Glutei
# S2	- Flexion of knee & extension of hip	-Hamstrings
# S3,4,5	- Anal contraction	-Anal & Perianal muscles

2) Sensory:

Pain and **parasthesia** in the area supplied by the affected root→ then loss of the superficial and deep sensations in the area supplied by the affected root.

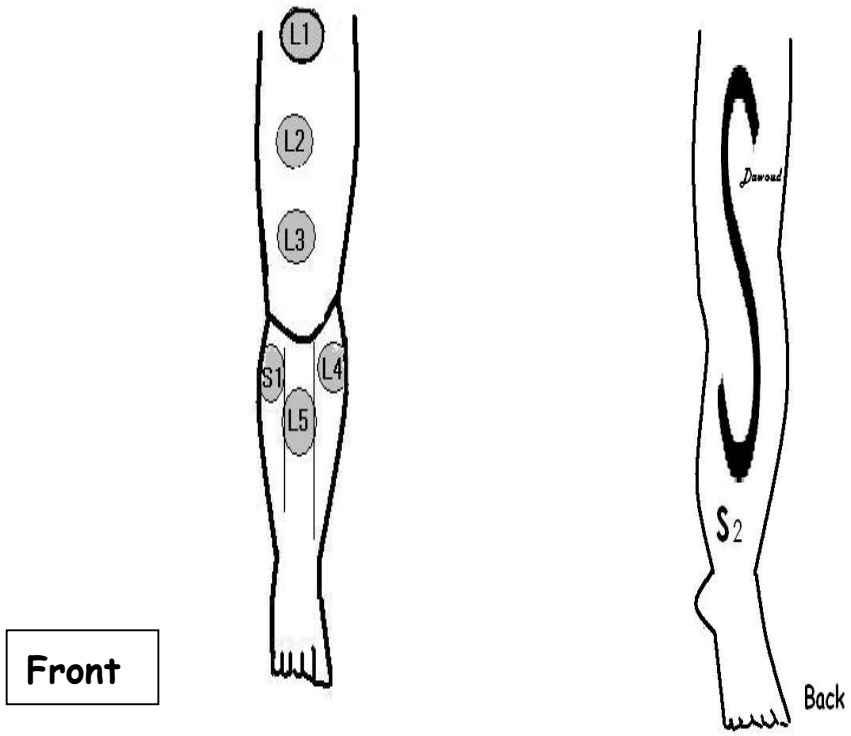
- **L1**→ Upper 1/3 of front of thigh.
- **L2**→ Middle 1/3
- **L3**→Lower 1/3
- **L4**→Anterolat. Aspect of the thigh , front of knee ,Anteromedial aspect of the leg & medial aspect of dorsum of foot and big toe
- **L5**→ Middle 1/3 of front of leg , dorsum of foot & middle 3 toes
- **S1**→ Posterolat. Aspect of thigh and leg , lat. 1/3 of dorsum & little toe
- **S2**→ Back of thigh and leg & sole of foot
- **S3,4,5**→Anal and perianal &gluteal region (Saddle shaped area)

Conus:

- ☑ Early autonomic bladder and fecal incontinence
- ☑ Impotence
- ☑ Impairment of sensations in saddle shaped area

Epiconus:

- ☑ Weakness or paralysis in muscles supplied by L 4,5 & S1,2
- ☑ Ankle reflexes absent BUT Knee reflexes are intact
- ☑ Sensory loss from L4 to S2
- ☑ Bladder disturbance **(***may occur in the form of pericipetancy* **)**



3) Bladder manifestations:

See neurogenic bladder

Neurogenic Bladder

Definition:

Sensory , Motor and Autonomic manifestations due to impaired nerve supply of urinary bladder.

Control of act of micturition:

- The wall of urinary bladder contain stretch and chemical receptors which are stimulated by fullness of bladder and acidity of urine .
- Efferent impulses are carried by S2,3,4 sensory parasympathetic fibers to 2nd ,3rd and 4th sacral segments of spinal cord .
- Efferent impulses carried by S2,3,4 motor parasympathetic fibers lead to contraction of the wall and relaxation of the sphincter → Evacuation (in infant)
- The Afferent impulses reaching the sacral part of the cord ascend in the post. Column → Brain where sensation of full bladder is perceived.
- Efferent impulses descend from a special bladder cortical center for the conscious control of micturition.

This center → *on the medial surface of cerebral hemisphere in front of foot area.*
→ *is developed with myelination of the pyramidal tract after the age one year.*

- Efferent impulses (inhibitory impulses)descend from this center to 2nd,3rd and 4th sacral segment through pyramidal tract.
- Micturition takes place normally when this inhibitory impulses are released

Supraspinal inhibitory impulses are absent in infants below one year due to Absence of myelination of pyramidal tract and immaturity of the cortical bladder center.

Lesions:

1) Afferent lesion :

Over urinary bladder filling with urine →gradual enlargement
(pr. of urine > pr. of sphincter)→ Dripping ## *Sensory atonic bladder* ##

2) Efferent lesion:

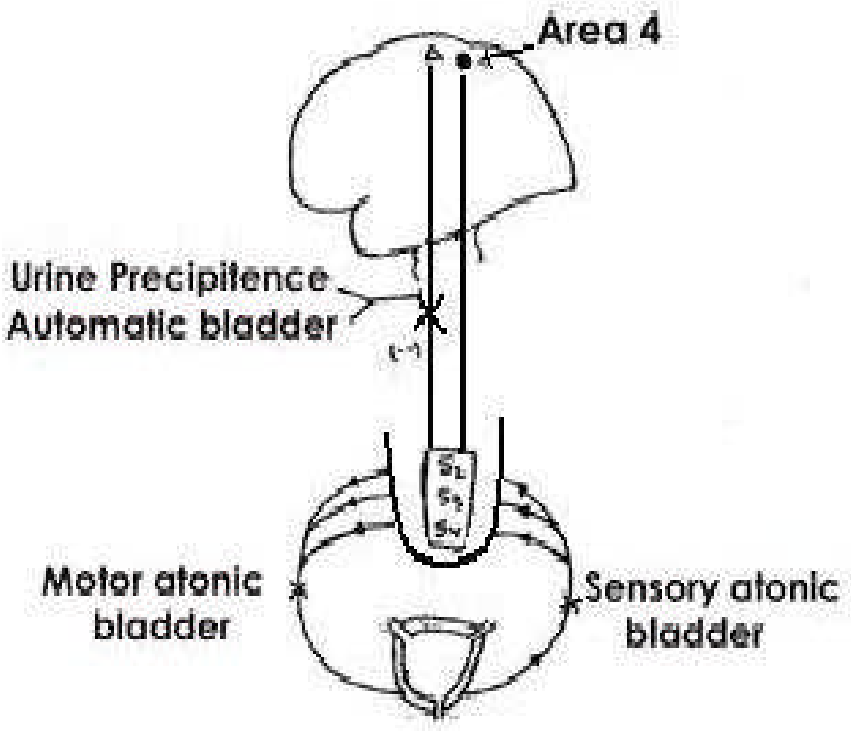
Mild urinary bladder filling with urine → Inability to evacuate voluntarily → Cauterization. ## *Motor atonic bladder* ##

3) Center lesion:

NO nerve supply to urinary bladder → evacuation by myogenic contraction
Autonomic bladder ## (**I**nvoluntary & **I**rregular & **I**ncomplete.)

4) Pyramidal lesion:

- ☑ **Acute lesion:** Retension of urine
- ☑ **Gradual lesion:**
  - **a) *Complete*:** ## *Automatic bladder* ## (involuntary & regular & complete)
  - **b) *Partial:*** Precipitancy of urine.



Peripheral neuropathy

Definition: Degeneration of peripheral nerves.

Classifecation:

1) Anatomical
   A. **Mononeuropathy**, affecting ONE nerve in ONE limb.
   B. **Mononeuropathy Multiplex**, affecting > one nerve in ONE limb
   C. **Polyneuropathy** ,affecting pr. Nerves of ALL limb
2) Atiological
   A. **Mononeuropathy**,(DM.&Leprosy &Polyartheritis Nodosa&Siatica)
   B. **Mononeuropathy Multiplex**, (DM.&Leprosy &Polyartheritis Nodosa&Sarcoidosis&Amylodosis)
   C. **Polyneuropathy** ,(Congenital& Acquired)

Manifestations:

- ***MOTOR:***
  1. Paralysis of muscles with LMNL (and mention its 5criteria)
  2. Bilateral and Symmetrical *as it is systemic*
  3. Affect distal muscles > Proximal
  4. Affect extensors > flexors
  5. Affect LL > UL
  6. Affect ankle reflex > knee reflex
  7. Affect **1673** nerves

N.B: the more the length of the nerve, the more ↓ its nutritional supply from the cell,
The more weakness and motor manifestations.

- ***SENSORY:***
  1. Glove and stock hyposthesia.
  2.

	Superficial Sensations	*Deep Sensations*
components	*-Pain - Touch -Temp.*	*-Muscles*
Irritation	*- Pain - Parasthesia*	*-Tender muscle*
Destruction	*-Lost superficial sensations*	*-Lost deep reflex - Sensory ataxia*

Autonomic: See Diabetic neuropathy.(*Endocrine book*)

Classification or Etiology of Polyneuropathy

CONGENITEAL

- **Proneal muscle atrophy**
  - # Inverted champine bottle appearance.
  - # Sever muscle wasting with mild weakness.
- **Refsum's disease**

* decrease in hydroxylase enz.→ increase **in phytanic acid→3S**

1. Cerebellum: ATAXIA
2. Skletal deformity
3. Special senses: BLINDNESS&DEAFNESS

***ttt.** *Plasma Phoresis*

AQUIRED

3 I & 2 T & MEN

1. **Metabolic:** DM. & Any failure { DM. affect **3rd & 4th & 6th** cranial Nerves} MCQ
2. **Endocrine:** all
3. **Nutrition:** vit.B complex deficiency
4. **Toxins:** ⇨ Endo.(LCf.& RF.)
   ⇨ Exo. (Heavy metals)
5. **Tumor:** Paramalignant $.
6. **Iatrogenic**: I.N.H
7. **Infection**:
   - **Diphtheria**:**{** mainly **motor** & bilateral affection & **10th** cranial N. affection**}** MCQ
   - **Leprosy**: **{**mainly **sensory** & loss of all sensations **& 5th , 7th** cranial Ns. affection**}** MCQ

     ttt. 1-Dapson
     2-Rifampicin
     3-Cipa 1906
8. **Immune**: →Collagen disease

   →Guillan Barre $

   Ascending paralysis & Cytoalbuminous dissociation

   3- phases: 1 Viremia (FHMA)
   2 Latent (Asymptomatic)
   3 Paralytic {mainly motor &**7th, 10th cranial** Ns. Affection} MCQ

   ttt

   ☑Plasma Phoresis ☑ Cortisone
   ☑Immunoglobulin. ☑ O2 ventilation
   ☑ ttt. Of DM.

Myasthenia Gravis (M.G.)

Definition:

It's a *neuromascular disorder* characterized by *easy fatigability* of skeletal muscles , specially on repetition of movement.

Etiology:

Auto-immune disease in which there are ***A.Choline receptors antibodies***

80% of cases associated with thymic dysfunction
50% of cases associated with thyrotoxicosis

Clinical Picture: ♀ > ♂ ☺

➲ *Easy fatigability* **(periodic skeletal muscle weakness)** characterized by:

❶ Diurnal variation with worsening of symptoms in the after noon

❷ Descending march

- **Ocular manifestations:** Ptosis & Diplopia
- **Jaw muscles:** mouth hanging open
- **Bulbar manifestations:** dysarthria : Hoarseness of voice
- **Neck muscles:** tilting of head
- **Skeletal muscles of UL & LL:**
  - ☛ Proximal > distal : e.g ; Hair Combing.
  - ☛ UL > LL

❸ No Sensory loss.

❹ Associated thymic enlargement, hyperthyroidism.

Diagnosis:

1. *Clinical test:*

Induction of fatigue by repetition of movement :

- Forward arm abduction time
- Counting from 1 to 50 → dysarthria & nasal tone
- ***Walker's test***: rise BP>Systolic, then Ask the patient to do rapid repetitive movement with his hand till exhaustion occur and then releasing the cuff
  → Ptosis

2. *Pharmacological test:*

- ***Prostigmine test****:* (anticholinesterase)
  Improvement of Ptosis within **20-30** minutes.
- **Tensilon (edrophonium) test:**
  Improvement of Ptosis within **2** minutes.

3. *Serological:*
   - Anti-acetylcholine Receptor (Anti-AchR) Antibodies
     Is **+ve** in more than **80%**of cases
4. *Muscle Biopsy &EMG.*
5. *X-ray chest to detect thymic enlargement*
6. *Thyroid function tests*.

Treatment:

➲ Medical

❶ **Prostigmine**: tab. 15mg & ampoule 0.5 mg
Onset: after 1/2hs. &duration : 3hs

❷ **Pyridostigmine:** tab.60mg & ampoule 1mg
Onset: after 2 hs. &duration : 8hs

So, we can combine Prostagmine and Pyridostigmine and give them to the patient every 8 hours

❸ **Tensilon (**edrophonium**):** IV. Only in myasthenic crisis

❹ **Cortisone:** Prednisone 60mg/day then 10mg/day maintenance dose

❺ **Immuno suppressive:** Azithioprine or Cyclophosphamide

➲ Plasma phoresis

➲ Surgical →Thymectomy.

Types of Myasthenia:

1. **1ry. Myasthenia:** Discussed before
2. **Neonatal Myasthena:**
   - It occur in **20%** of babies born to myasthenic mother.
   - Symptoms usually start within **3 days** of birth
   - *c/p.* poor sucking & Poor crying.
   - *ttt*. Prostagmine (o.o5 -0.1mg) iv every 4 hours
3. **Eaton-Lambert Myasthenic $:**
   - It may occur as paramalignant manifestations e.g **Small cell carcinoma**
   - It may also occur with other Auto-immune diseases
     An IgG antibodies bind to nerve terminals→ ↓↓ release of A.choline
   - *Invest.* EMG &X-ray chest for bronchogenic carcinoma
   - *ttt*. Pyridostegmine - Guanidine – cortisone
4. **Myasthenic crisis:**
   - paralysis of ***respiratory*** or ***swallowing*** muscles may occur.
   - *ttt*. Edratropium & plasma phoresis & Mechanical ventilation.

Wasting of small muscles of the hand

The small muscles of the hand are supplied by C_8 and T_1 spinal segment

Median nerve palsy usually due to carpal tunnel syndrome.	*Suggested by:* wasting of thenar eminence. Weakness of thumb flexion, abduction and opposition. **Unable** to lift thumb with palm upwards but **able** to press with index finger. Loss of sensation over palmar aspect of radial 3.5 fingers of hand. *Confirmed by :* nerve conduction study results.
Ulnar nerve lesion	*Suggested by:* wasting of hypothenar. Able to lift thumb with palm upwards but unable to press with index finger. Weakness of finger abduction and adduction. Loss of sensation in ulnar aspect 1.5 fingers of hand. *Confirmed by*: nerve conduction study results.
T1 lesion: anterior horn cell or root lesion	*Suggested by:* wasting of all small muscles of hand. Unable to lift thumb with palm upwards , unable to press with index finger. *Confirmed by:* nerve conduction study results. MRI scan appearance around T1 level.
Motor neurone disease	*Suggested by:* **signs of T1 lesion**, prominent fasciculation, spastic paraparesis, wasted fasciculating tongue, no sensory signs. *Confirmed by:* absence of structural abnormality on MRI scan appearance.
Syringomyelia	*Suggested by:* **signs of T1 lesion**, fasciculation not prominent, burn scars, dissociated sensory loss, Horner's syndrome, nystagmus. History over months to years. *Confirmed by:* MRI scan appearances.

Any prolonged systemic illness	*Suggested by:* global muscle wasting, general weight loss. *Confirmed by:* improvement in muscle wasting if primary disease treatable.
Cervical spondylosis	*Suggested by:* **signs of T1 lesion**, neck pain and stiffness, and referred pain. *Confirmed by:* MRI
Tumor compressing nerve root	*Suggested by:* **signs of T1 lesion**, referred pain. Progressing over months. *Confirmed by:* MRI
Brachial plexus lesion	*Suggested by:* **signs of T1 lesion** and history of trauma to shoulder area or birth injury. *Confirmed by:* nerve conduction study results.
Cervical rib	*Suggested by:* **signs of T1 lesion** aggravated by movement or posture. *Confirmed by:* X-ray . Relief by surgery.
Pancoast tumour	*Suggested by:* **signs of T1 lesion**, Horner's syndrome, features of lung cancer (clubbing, chest signs, lymph nodes etc.). *Confirmed by:* CXR and CT scan appearances.

A Physician without differential diagnosis is like a Soldier without his gun, like a Clown without his fun.

AL-SAYED DAWOUD

Subarachnoid Hemorrhage

Definition: hemorrhage in the S.A. space

Etiology:

1. **Aneurysm** (*congenital*) **85% → the most common cause** MCQ
2. **Angiomateous malformation 15%**
3. **H**ypertension
4. **H**emorrhagic disease
5. **T**umor
6. **T**rauma

Clinical Picture:

1) Pre rupture :



A. **T**ension Headache
B. **T**ransient any focal lesion (Cranial nerves & c.c.)
   # Compression on cranial nerves esp. 2,3,4,5,6 nerves
   # Aphasia, Monoplegia, Convulsions ……. and mention

2) At rupture :

A. Headache: **S**ever, **S**ustained, **S**plitting→typically called "the worst headache in my life"
B. Permanent any focal lesion (Cranial nerves & c.c.)
C. Blood in S.A space
   - ✓ ↑ **I.C.T.** (headache , Blurring of vision , Vomiting)
   - ✓ **Fever** due to chemical meningitis by blood products.
   - ✓ **Meningeal Irritation**…….. NECK RIGIDITY (see meningitis)

   So, the condition should be differentiated from meningitis by Lumber Puncture
   1. Increased Pressure
   2. Normal culture
   3. Increased Protein
   4. Bloody

	Subarachnoid Blood	*Post Traumatic Blood*
1. *Samples* 2. *Blood Clotting* 3. *I.C.T.* 4. *Aspect*	▪ *Constant in all samples* ▪ *Absent* ▪ *Increased* ▪ *xanthochromia*	▪ *Decreased in successive samples* ▪ *Occur* ▪ *Normal* ▪ *Clear*

3) After rupture:

Reflex vasospasm in cerebral blood vessels

Investigations:

1) Lumbar puncture
2) C.T
3) MRI
4) Angiography

Comlications:

- Sudden death
- Rebleeding: if the aneurysm is not repaired promptly
- Cerebral vasospasm
- Hydrocephalus
- Seizures: 15 – 90 % the patients

Prognosis:

- 50% :die , 2/3 of them die within 24hs
- 20% :die within 6 months due to **Rebleeding**
- 20% :hemiplegia &epilepsy
- 10% : symptom free

Treatment:

- ✓ **MEDICAL**
  1) ***Care of comatosed:***
     - Absolute bed rest & Avoid straining (5b)
  2) ***Symptomatic ttt.***
     - General dehydrating measures
     - Antiemetics
     - Antipyretics
     - Morphine
     - Muscle relaxant
  3) ***Blood Pressure***
     - ***Aim*;** to maintain systolic blood pressure **<** 100mmHg
     - Nefedipine (CCB)
  4) ***Antifibrinolytics***
     - Epsilon Amino Caproic Acid
- ✓ **SURGICAL**
  - To prevent rebleeding
  - Carotid ligation
  - Excision of the aneurysmal sac
- ✓ **INTERVENTION**
  - Catheterization in femoral artery ⇨Aorta ⇨CCA. ⇨ ICA. ⟹ Aneurysm.

Ataxia

Definition: Incoordination of voluntary movements with **or** without disequilibrium
In absence of paralysis or paresis.

Types:

(1) Cerebellar Ataxia

Etiology:

1. **Heridofamilial** ➔ Friedrich's ataxia & Marie's ataxia
2. **Vascular** ➔ PICA & AICA
3. **Toxic** ➔ Alcohol & Barbiturates
4. **Neoplastic** ➔ Medulloblastoma

Clinical picture:

- **Ataxia**
  1. **Limb:**
     - **a. kinetic tremors and dysmetria , detected by:**
       - Finger to nose test
       - Finger to finger test
       - Heel to knee test
       - Buttoning and unbuttoning
     - **b. Adiadokinesia**
     - **c. Rebound phenomenon**
  2. **Eye:** Nystagmus
  3. **Tongue:** Stacatto Speech
- **Hypotonia** marked with acute lesion
- **Disequilibrium** ➔ Wide Gate
  ➔ Drunken Gate
  ➔ Zigzag Gate (if bilateral)

	Friedrich's ataxia	Marie's ataxia
Degeneration of :	Cerebellum and 3p ☞ Pyramidal tract ☞ Peripheral nerves ☞ Posterior column	Cerebellum and Pyramidal tract
Features:	+ve. Babiniski Sign Hypotonia & Hyporeflexia	+ve. Babiniski Sign Hypertonia & Hyperreflexia
Association:	Skeletal deformities Cardiomyopathy	Mental Retardation Optic neuritis

(2) Sensory ataxia

Definition: Ataxia due to loss of deep sensations

Etiology:

1. **P**eripheral Nerves: Peripheral neuropathy
2. **P**osterior column: SCD
3. **P**osterior root: Tabes dorsalis
4. Thalamus : Thalamic syndrome
5. Cortical sensory area :Parital lobe lesion

Clinical picture:

- Clinical picture of ataxia **(***on eye closure***)**
- Loss of deep sensation: loss of vibration sense & loss of position and joint sense
- Stamping gait
- Romberg's Sign **(Inability to maintain the upright position with eye closed)**

(3) Vestibular ataxia

Definition: Lesion of vestibular division of 8th cr.nerves

Etiology: Menier's disease & labrynithitis

Clinical picture:

- Vertigo &Tinnitus
- Nustagmus & disequilibrium.

Extra Pyramidal System

Definition:

Any fibers that can influence movement **Without** passing in the pyramidal system.

Anatomy: see the figure

Function:

1. *Filtration of motor impulses from area* **4**
2. *Regulation of muscle tone*
   - **A**cetyl Choline → **A**ctivate **(** ↑ Muscle tone**)**
   - **D**opamine → **D**ecrease **(** ↓ Muscle tone**)**
3. *Emotional, associative and automatic movements.*

So, any lesion in the extra pyramidal System is characterized by:

❶ Involuntary movement: (*8 charecters*)
- ⇨ Involuntary & static & ↑↑ by stress & ↓↓ with sleep.
- ⇨ In parkinsonism: (Regular & Rhythmic & Distal> Proximal & Money count)
- ⇨ In Chorea :(irregular & dysrhythmic & proximal & purposeless)

❷ Muscle tone : all increase muscle tone *Except* chorea

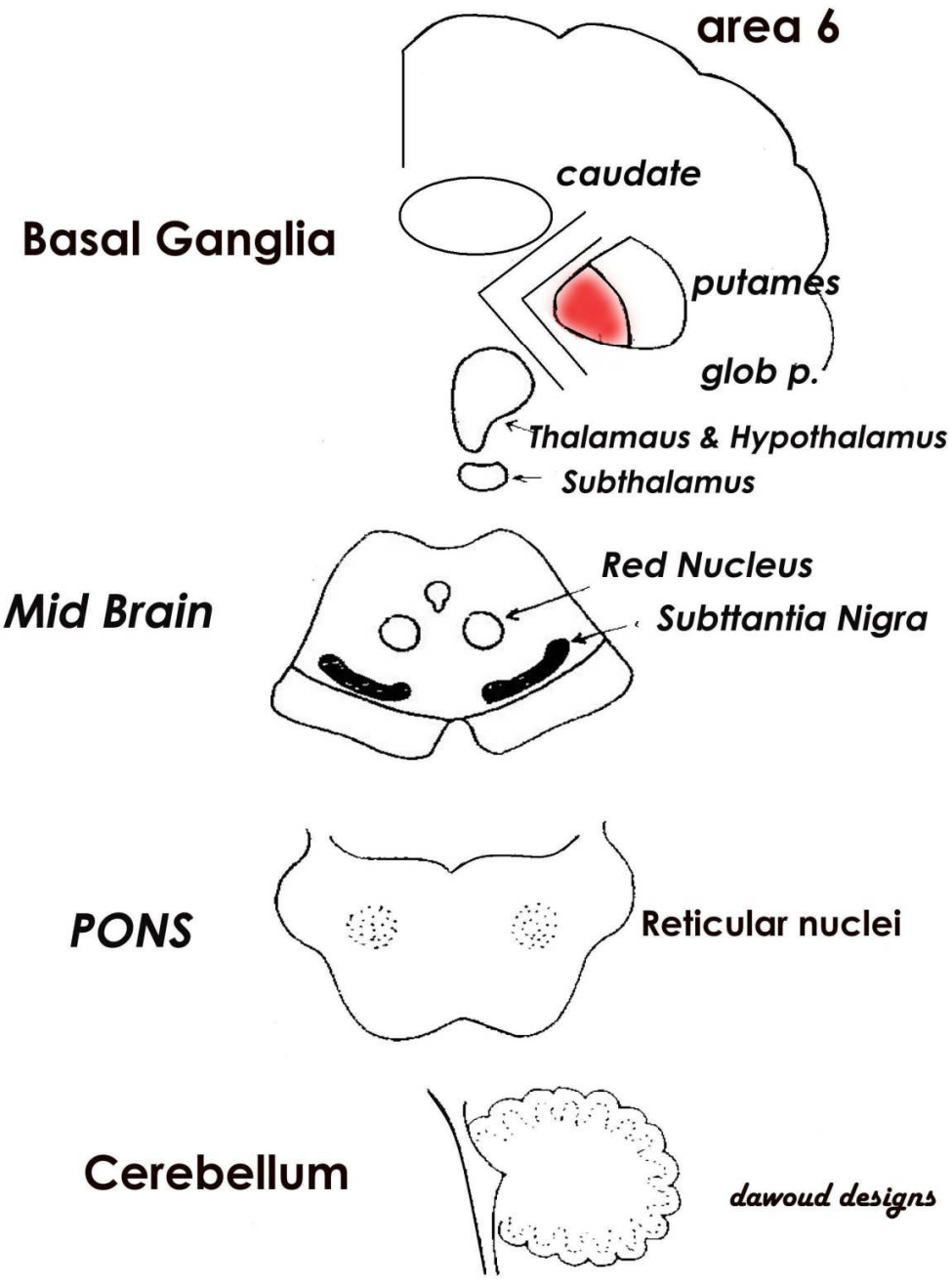
❸ Emotions:
- ⇨ Parkinsonism → bradykinesia
- ⇨ Chorea → exaggerated (emotional instability)

Addiction-Free Nation Program should consider **in capsule series** its **1st** priority, as it has been proved that almost all internal medicine seekers are addicted to it.

Alsayed Dawoud
Kasr-Alainy School of Medicine

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the Extrapyramidal System



In Capsule Series

Parkinsonism

Neurochemichal basis:

↑ Dopamine in the basal ganglia and substantia nigra
↓ Acetyl Choline

Etiology

1. Idiopathic (paralysis agitans) → *The commonest type*
   - ☞ Degenerative type of the disease
2. Post Encephalitis
3. Cerebral Atherosclerosis

others:

4. Wilson's disease
5. **T**oxic : Co & Mg
6. **T**umor
7. **T**rauma : Rare

Clinical Picture: ***B.Ri.T.ch man***

Triad of: 1-**T**remors
2-**Ri**gidity
3-**B**rady kinesia

1-Tremors:

8 (**hyperkinesia**)

4

-Involuntary
-**S**tatic
- ↑↑ by **S**tress
- ↓↓ by **S**leep

4

-Rhythmic
-Regular
-Distal > Proximal
-Money count(4-8/sec.)

D.D. of Tremors:

1) Parkinsonism
2) Senile tremor
3) Familial tremor
4) Hysterical tremor
5) Cerebellar tremor
6) Thyrotoxicosis
7) Flapping tremor
8) Alcohol & Amphetamine

2-Rigidity:

	Rigidity	Spasticity
1-Site of lesion	-Extra pyramidal tract	-Pyramidal tract
2-Distribution	-proximal >distal -flexors of UL&LL	-distal > prximal -flexors of UL& extensors of LL
3-Charactres	- lead pipe or cogwheel	-clasp kife.
4-Deep reflexes	-heporeflexia	-hyper reflexia

D.D. of Hypertonia

1-Pyramidal lesions (UMNL): Claps knife type

2-Extra pyramidal lesion : Parkinsonism (Lead pipe or Cog weeel)

3-Hysterical

4-Toxic: Resirpine , Co : Cog weel rigidity.

5- Meningitis

6-Myotonia

3-Brady Kinesia:

1-Monotonus speech

2-Mask face

3-Lack of blinking

4-No swinging of arm

Difference between the most common 3 causes of Parkinsonism

	diopathic	Encephalitis	Thrombosis
1-Age	-Around 50	- <40	- 40-60 years
2-Onset	-Gradual	-Acute	-Gradual
3-Course	-Progressive	-Regressive	-Remission &exacerbation
4-Tremors & rigidity	-Tremors> regidity	-Regidity > tremors	-Rigidity> tremors
5-Assosciation	- NONE	-Occulogyric Crisis -Pyramidal signs: * Scuating * Sialorrhea * D.I. * Obesity * Impotence -History of Encephalitis may be obtained	-Hypertension -D.M. -Coronary H.D. -Pyramidal manifestations

Investigations: No specific investigations ☺

-Routine investigations (MRI & CT& Tests for Wilson's D. e.g. caeruloplasmin and serum cooper)

Treatment:

1. **Anticholinergic drugs**
   - benzatropin (Congentin) & tremaril
     - # ***Side effects :***
       - Retention of urine .
       - Tachycardia.
       - Dryness of mouth.
2. **L – dopa**
   - It is transformed to dopamine peripherally which can't pass BBB.
     - # ***Side effects :***
       - psychosis
       - palpitation
       - GIT upset
       - Chorea→ *On and Off phenomenon*
3. **Sinement**
   - (L.dopa + Carbidopa) inhibit the peripheral decarboxylation of L-dopa into dopamine So L – dopa will pass through BBB.
4. **Amantadin**
   - uptake of dopamine by the neurons and used as adjuvant therapy.
5. **Ms relaxant** and **B.B**

Chorea

Definition:

Involuntary,static,irregular,dysrythmic,sudden,jerky,pseudopurposive movement of any part of the body due to lesion in caudate nucleus.

♀>♂ ☺ (↑ dopamine - ↓ Ac.choline)

Etiology:

1. **Idiopathic** (*Senile chorea*)
2. **Encephalitis** (*post encephalitic chorea*)
3. **Vascular** (*Hemiballismus*)
4. **Toxic** (*chorea gravidarum*)
5. **Heridofamilial** (*Huntington's chorea*)
6. **Autoimmune** (*Rheumatic chorea*)

Rheumatic chorea {*Sydenham's chorea*}

c/p:

I. Movement:

- **4** characters ***as*** Parkinsonism
  {Involuntary & **s**tatic & ↑↑ by **s**tress & ↓↓ with **s**leep}
- **4** characters ***opposite*** Parkinsonism
  {Irregular & dysrhythmic & **p**roximal & **p**urposeless}

II. Hypotonia:

- Dancing gait
- Grimacing smile
- No tongue protrusion without teeth support
- Arm deviation down when elevated
- Scaphoid hand → flexion at the wrist
  → Over extension of metacarpophalyngeal and interphalengeal joints

III. ↑ Emotions:

Emotionally instability as sudden crying or laughter

Clinical varieties of Rh. Chorea

- ⇨ Chorea **gravis** (*disorder of Sleep, Speech & Swallowing*)
- ⇨ Chorea **mollis** (*flaccid paralysis*)
- ⇨ Chorea **mania** (*instable emotions*)

Treatment:

☑ **ttt. Of Rheumatic fever**

- Complete bed rest
- Acetyl salicylic acid *6-8mg/day*
- Prednisone in rheumatic activity

☑ **Dopamine antagonists**

- Hallopridol & Reserpine

☠ Huntington's chorea ☠

- Autosomal dominant → chromosome **4** MCQ
- Middle age
- Gradual onset & Progressive course→ Fatal type of chorea
- Due to degeneration of: #→***Basal ganglia***

{Gross movement with interference with feeding and walking}

#→**Cerebral cortex** (***prefrontal area***)

{Change in behavior and mentality}→ Dementia is prominent

☠Hemiballismus chorea☠

- It is a form of forceful, flinging, high-amplitude, coarse, chorea.
- Involuntary movements usually affect only one side of the body
- Due to lesion involving the contralateral **subthalamic nucleus**(**STN**) MCQ
- Subside spontaneously within several weeks

Athetosis

1) Involuntary movements: **(8** *criteria***)**



- ⇨ **I**nvoluntary & **S**tatic & ↑ by **S**tress & ↓ by **S**leep
- ⇨ Irregular
- ⇨ Dysrhythmic
- ⇨ Distal > Proximal
- ⇨ Snake like movement

2) Hypertonia: *during the movement*

3) Facial grimacing & *dysarthria may occur*

Dystonia

1) Involuntary movements

- ⇨ **I**nvoluntary & **S**tatic & ↑ by **S**tress & ↓ by **S**leep
- ⇨ Very Slow twisting like movements
- ⇨ Affects neck & trunk & proximal extremities

2) hypertonia : *during the movement*

3) No emotional manifestations MCQ

- ⇨ -ve reflexes & Normal sensations, mentality & Speech

DD. of involuntary movements:

1. Epilepsy→Convulsions
2. Extra pyramidal lesion→ Parkinsonism & Chorea &Athetosis &Dystonia
3. LMNL→ Irritative lesion
4. Psychomotor movements→ Tics
5. Tremors

Motor Tics

Involuntary, Abrupt, Sudden, Isolated, Brief movements

Types:

Simple: e.g→ Eye blinking

Complex: e.g→ kicking & pelvic gyration

Headache

Definition: Pain over the level of the eye brows to the sub-occipital area.

Sensitive structures in the skull

1. All the tissue covering the bone except the hair
2. Cerebral arteries
3. Venous sinuses
4. Cranial nerves: **5,9,10** & upper **3** cervical nerves
5. Meninges: especially the basal

N.B: *the Brain itself is insensitive*

Etiology

1) Vascular headache : (*V.D.*)

- Migraine
- **H**ypertension & **H**ypoxia & **H**ypoglycemia
- **A**rteritis & **A**neurysm
- Toxic: → Caffeine withdrawal & Alcohol
  → renal and hepatic failure

2) Traction Headache: (*Space occupying lesion*)

- due to stretch of Meninges as in brain tumors

3) Tension Headache: (*Psychogenic headache*)

- Cap-like constriction (Ache pain)
- Not localized & Not throbbing & Don't increase with Straining
- Usually associated with depression, neurosis & stress

4) Meningeal irritation:

- Meningitis
- Subarachnoid hemorrhage

5) Muscle contraction headache:

- Prolonged contraction of the muscles of the head and neck **e.g**: prolonged driving

6) Neuritis and neuralgia:

- Of the sensory nerves of the scalp **e.g.**: trigeminal neuralgia

7) Referred headache: *may be very sever*

- Eye: iritis, glaucoma
- Ear: middle ear diseases
- Nasal sinuses diseases
- Teeth diseases and Tongue diseases

Migraine

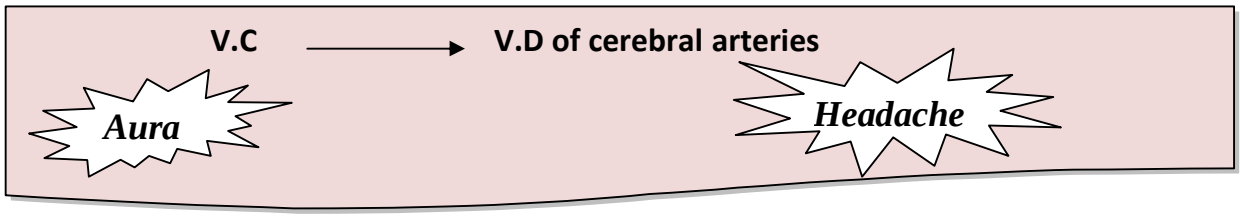
Definition:

Paroxysmal attacks of throbbing headache usually unilateral rarely preceded by aura.

Etiology: the exact cause is unknown

- ♀ > ♂ ☺
- Genetic factor may play a role ***70% Heridofamilial*** MCQ
- Type A personality

Mechanism: ischemia followed by V.D



Theories:

- **Serotonin theory:** initial increase in serotonin → **V.C** of cerebral arteries
  Then, degeneration of serotonin → **V.D** of cerebral arteries
  - ☞ Serotonin receptors in the other parts of the body are affected **{***nausea* & *vomiting***}**
- **Hypoxic theory:** **V.C** of cerebral arteries followed by **reactive V.D**

Clinical picture: (4 stages)

❶ **Prodroma:** changes in mood or appetite days before the attack.

❷ **Aura:** immediately before the attack

2 V (**V**isual hallucination & **V**ertigo)
2 P (**P**arasthesia & **P**aresis for about **5** minutes)

❸ **Headache:**

1. **Site**: usually unilateral (starts in the temple or around the eye)
2. **Type:** throbbing pain (*very sever*)
3. **Duration:** lasting **4** to **72** hours
4. **Increased by:** exercise, light & menstruation
5. **Decreased by:** sleep
6. **Association:** nausea and vomiting

❹ **Post headache phase:** polyuria

Types of migraine:

1. **Classic migraine**: migraine with aura **15%** only MCQ
2. **Common migraine:** without aura **85%**
3. **Variants:** according to aura **{**Hemiplegic or Ophthalmoplegic migraine **}**
4. **Cluster headache :** Horton's syndrome = **Migranous Neuralgia**

☠ *Migranous Neuralgia* ☠

- ***Frequent attacks of headache which is usually associated with conjunctival injection, lacrimation, rhinorrhea, flushing and sweating of the affected side of the face.***
- ♂ **>** ♀ ***middle age***
- ↑ ***By histamine, alcohol or heat***
- ***ttt.O2 inhalation*** 100% & ***Ergotamine***

Treatment:

☑ During the attack (**4**)

1. Rest in quite room
2. Analgesics: aspirin or paracetamol in mild attack
3. **Ergotamine** (serotonin agonist)
   - ☞ Oral, sublingual or nasal spray
   - ☞ Migranil® **or** Imigran®
4. Antiemetics

☑ Between the attack (**4**) ***A,B,C,D***

1. **A**void precipitating factors
2. **B**.Blockers: prevent V.D
3. **C**CB: flunarizine HCL
4. **D**eseril: serotonin antagonist
   - **S.E:** retroperitoneal fibrosis MCQ

Status **M**igrainousus

Def.: migraine attacks that persist for longer than 72 hours despite treatment

C/p: ⇨ **headache-free periods for less than 4 hours may occur**
⇨ **Usually associated with prolonged use of analgesics**

TTT. → **Hospitalization**
→ **ttt with detoxification**

Myopathies

Definition:

Degeneration of the muscle fibers with normal peripheral and central nervous system.

Classification of myopathies:

I. ***Idiopathic:*** genetically determined progressive muscular dystrophy
II. ***Inflammatory:*** polymyositis (*see rheumatology*)
III. ***Iatrogenic:*** Cortisone
IV. ***Metabolic:*** **Hypo** or **Hyper**kalemia
V. ***Endocrine:*** **Hypo** or **Hyper**thyroidism

C/P of progressive muscular dystrophy:

There are many types of progressive muscular dystrophy BUT, all the types share in the following criteria.

5 facts

- Age of onset usually **below 10 years**
- **+ve** family history
- Paresis of muscles of **LMNL**
- **Symmetrical** and Bilateral (systemic disease)
- Affect proximal muscles > distal muscles

5 Examples

- Winging of the scapula
- Post-belly abdomen: due to weakness of abdominal muscles
- **Lordosis**: due to weak trunk muscles
- **Waddling gait**: due to weakness of pelvic muscles.
- **Gower's sign**: climbing on getting up from the ground

NO

- ☒ **No** sensory loss
- ☒ **No** bladder manifestations
- ☒ **No** affection of sternomastoid muscle.

Clinical types of progressive muscular dystrophy:

Shoulder girdle types:
- Scapula-humeral (Erb's)
- Facio-scapulo-humeral

Pelvic girdle types:
- Pseudo-hypertrophic: **Duchenne & Becker** types

Other types:
- ***Distal type:*** onset distally, proximal involvement is later
- ***Ocular type:*** ext. ocular muscles, may also face, neck &arms
- ***Oculo-pharyngeal:*** as in ocular but with dysphagia

Duchenne	Becker
Malignant	Benign
Age of onset 1-5 years	5-25 years
Pelvic then shoulder, later limbs &respiratory ms.	Pelvic then shoulder *BUT* No respiratory muscle affection
Rapid progressive	Slow progressive
Degeneration of cardiac muscle	No affection of cardiac muscle
Death within about 15y' of onset	May have normal life span

Investigations:

1) ***Estimation of creatine and creatinine in urine***
   - Normally, no creatine in the urine of adults and creatinine is present
   - Because of creatine is metabolized to creatinine in the muscles
   - BUT, in myopathy; diseased muscle can't do it.
   - So,↑↑ creatine in urine and ↓↓ Creatinine in urine MCQ
2) ***Serum enzymes:*** CPK↑↑
3) ***EMG:*** characteristic
4) ***Biopsy***: Fibrous and Fatty tissue

Treatment: { No specific ttt. Management is supportive }

- ***Genetic counseling***: to detect the carrier
- ***Vitamins:*** especially vitamin **E** (↑ creatinine in the body)
- ***ATP:*** 25mg/day **I.M**
- ***Prophylaxis:*** against **DVT** and **Pneumonia**
- ***Physiotherapy***

Causes of death in muscle dystrophies:☠☠

- Respiratory failure
- Pneumonia
- Cardiomyopathy→ in Duchenne

Multiple Sclerosis

Definition:

This is a disease of unknown cause characterized by affection of white matter of the central Nervous system resulting in degeneration of its myelin sheath.

Etiology:

- Autoimmune: as CSF show ↑ immunoglobulins
- virus (*controversy*)

Clinical picture:

⇨ age → 20- 50 years

⇨ ♀ : ♂ → 3: 2

⇨ Onset → acute or gradual

⇨ Course →Remission & exacerbation

Mentality: Euphoric - emotional instability

Speech: Slurred , Staccato or Scanned

Cr. Nerve:

1. Optic→ **Optic neuritis**
2. Facial→ **LMNL & UMNL**
3. Oculomotor→ **Diplopia & Ophthalmoplegia**

Motor: ***usually UMNL*** MCQ☑

1. paraplagic ✷ Quadriplagic
2. monoplagic ✷ hemiplegic

Sensory: Superficial & deep sensory loss ***+ve L'hermite's sign***

Cerebellar: tremors & Nystagmus

Sphincteric disturbance & Impotence☠☺

Investigations:

I. CSF Examinations: ***Gammaglobulin IgG*** & ***Oligoclonal bands***

II. Visual evoked potential to detect early affection of ***Optic Nerve***.

III. MRI: more sensitive during exacerbation

IV. C.T. Scan → hypodense area in acute stage

Treatment of multiple sclesosis:

- *Treatment of acute exacerbations:*
  - ***Pulse steroid*** therapy by ***methyl prednisolone*** 1 gm/D for 3 to 5 days.
- *Prevention of relapse:*
  - Interferone.
  - Azathioprine.
- *Symptomatic treatment:*
  - Fatigue: give flouxetine.
  - Spasticity: give muscle relaxant.
  - Bladder: give anticholinestrase.
  - Ataxia or tremors: give Buspirone

Alsayed Dawoud

Epilepsy

Definition:

It is a paroxysmal disorder characterized by recurrent transient disturbances of the brain functions (motor ,sensory, psychic or autonomic) associated with EEG changes with or without disturbed consciousness.

Æ:

1) **Idiopathic :** { the commonest }

➲ +ve FH. , start in 1st decade

a) General mal epilepsy

b) Petit mal epilepsy

c) Myoclonic epilepsy

2) **Symptomatic:**

➲ *Epilepsy is only a symptom of underlying brain lesion.*

I. <u>Local:</u>

- **Congenital :**ccerebral palsy
- **Traumatic :**birth trauma ,head injuries
- **Inflammatory:** meningitis encephalitis brain abscess neuro syphilis
- **Vascular :** hge ,thrombosis embolism
- **↑ICT :** tumor brain abscess
- **Degenerative :**pre-senile dementia

II. <u>General causes</u> è 2ry effect on brain

- ☺ **Toxic:** alcohol ,botulism CO2 ,cyanide tetanus
- ☺ **Hypoxic:** pulmonary disease →↑↑RR→hyper ventilation →Co2wash→alcalosis→↓threshold of brain cells
- ☺ **Ischemic:** adam stoke's attacks , carotid sinus syndrome ,F4 ,anemia.
- ☺ **Metabolic:** hypo&hyperglycemia ,hyponatremia ,hypercalcaemia ,RF ,LCF ,HF ,Resp.F.

- ☺ **Endocrinal** : DM ,thyrotoxic crisis hypoparathyroidism
- ☺ **Hyperthermia** : sun stoke water intoxication high fever especially in children
- ☺ **Iatrogenic** amphetamine aminophylline ambilhar improper use of antiepileptics
- ☺ **Nutritional** pellagra Beri Beri GABA ↓↓
- ☺ **Hysterical**

Classifications:

I. **Partial seizures** : symptomatic 2ry
Start focally in one region of the brain
   a. Simple partial seizures with maintained consciousness
   b. Complex partial seizures with impaired consciousness
   c. Partial progressing to generalized tonic clonic convulsions

II. **Generalized seizures** idiopathic 1ry
Start in both cerebral hemispheres at the same time
   a. Petit mal typical &atypical
   b. Tonic clonic tonic clonic
   c. Myoclonic

Clinical picture:

1. Generalized:

a. **General mal epilepsy** old age

1. Pre-ictal :

Phase: aura

Good → warning sings for coming attack

Pad → 3-2 min before the attack

May be somatic, psychic and autonomic.

Gives an idea about the site of the attack

2. **Ictal :** 3phases seizures

i. Sudden loss of consciousness about 10 min
ii. Tonic phase 10sec with the onset of coma
- Rolling eyeball upward
- Clenching of teeth
- Extention of UL &LL
- Telting of head to one side
- Res. ms. Contraction →cyanosis
- Laryngeal ms contraction → epileptic cray

iii. Clonic phase 2-3min
- Repitation of contraction &relaxation
- Micturation
- Tongue biting

3. Post ictal:
Phase : <24h due to neuronal exhaustion
- ~ Fatigue
- ~ Headache
- ~ Sleepiness
- ~ Weakness
- ~ ***Todd's paralysis*** →transient paralysis following fit

➔Drug of choice carbamazepine _ tegretol _ phenytoin

b. **<u>Petit mal epilepsy</u>** ➔in children &improve at puberty
Interruption of stream of consciousness {absence seizures}→no aura
May be precipitated by hyperventilation or photic stimulation.

I. Typical :
Sudden loss of consciousness of short duration "few sec" è cessation of motor activity or speech è blank expressions of the face.

II. **Atypical**:
1) Pyknolepsy: high frequency 30-100/day for few sec
Staring look , not fall to the ground
2) A kinetic"atonic" :attacks of sever hypertonia
Fall to the ground without warning "no aura"
3) Myoclonic: sudden jerky movement of the hand &UL

c. <u>Myoclonic epilepsy :</u>

1. Simple myoclonus
2. È general mal epilepsy
3. È petit mal epilepsy
4. Juvenile myoclonic epilepsy :

   12-16 Ys –frequent jerks upon awaking &disappear later in morning
   May be accompanied è generalized tonic-clonic
   ttt è valpromate for life

➲ May occur in ms of face ,palate, extremities
➲ There is sudden shock –like involuntary movement

d. <u>Atonic "astatic" seizures</u>

✶ Start in children
✶ Attacks èout warning &for seconds
✶ Sudden loss of postural tone &fall to the ground no loss of consciousness
✶ Response to ttt is poor

2. Partial seizures

A. ***<u>Simple:</u>***

No loss of consciousness
according to the site of the excitatory focus in the brain

1. Sensory:

- **General: {**area **1,2,3** irritation } in the form of parasthesia involving one limb (focal) or **1\2** of the body sensory jacksonian fits
- **Special**
  - Visual hallucinations "formed –unformed "
  - Olfactory &gustatory hallucination "irritation of uncus "
  - Auditory hallucinations
  - Depersonalization –derealisation déjà-vu or Jamai-vu phenomena irritation of temporal lobe

2. **Motor focal :**

Movement of part of a limb or the whole limb
Motor Jacksonian fits: movement involving one side of the body spread in a march course "above down or down above.

B. ***Complex***

There is disturbed consciousness
The lesion in the temporal lobe.

1. ***Aura:*** olfactory &gustatory hallucination emotion &fear of elation Depersonalization –derealisation déjà-vu or jamai-vu phenomena
2. ***Absence*** the patient appears distant è starring eye &may not respond to questions
3. ***Automatism*** here may e a champing of teeth ,licking of lips or semi purposeful movements of limbs the patient may walk &leave the room may become violent
4. ***Amnesia*** for the attack

C. ***Partial seizures where*** simple or complex may progress to generalized tonic-clonic seizures

3. **2dry :** (symptomatic)

1. Sensory ➔ area1,2,3

- Affect part of area → pain &parasthesia part of the body e.g. hand
- Affect all of area →pain &parasthesia of ½ of the body{sensory jacksonian fit}
- Also visual, auditory or olfactory hallucinations may occur.

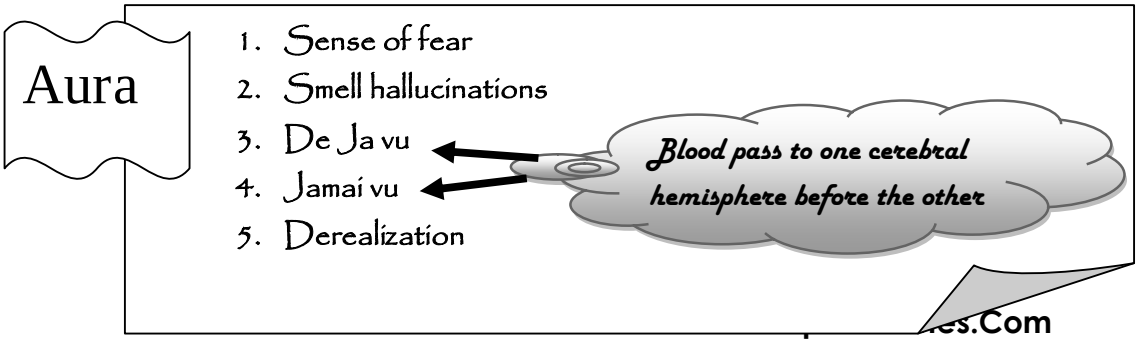
2. Motor➔ area4

- Affect part of the area→convulsions in part of body {motor jacksonian fit }
- Affect all of the area→convulsions in ½ of body {motor jacksonian fit }

N.B partial simple epilepsy =sensory +motor +no loss of consciousness

Psychic ➔ temporal lobe

- Psychosensory aura only
- Psychomotor 4A ☺
  - **Aura**
  - **Absence**
  - **Automatism**
  - **Amnesia**



Investigations:

Aim:

- to prove the condition
- to differentiate symptomatic from idiopathic causes

1. **EEG**
2. **X**-ray, **CT**, **MRI**
3. **CSF** examination
4. **Lab :** renal functions ,blood sugar , CBC, …..

Treatment:

I. General :

1. Modification of life :

   No swimming,
   No driving,
   Adjust the work of the patient to keep him safe away of accidents
2. Avoid ppt factors :
   - photic stimulation
   - missed drugs (bad ttt)
   - high sounds
3. diet :
   - ***No*** alcohol
   - ketogenic diet : fat induce acidosis
4. ttt of the cause

II. specific ttt :

⇨***Basic principles***⇦

1. Monotherapy :

   Is the best approach to newly diagnosed epileptics ,the drug of choice is selected according tp seizure type
2. Multiple drugs are more expensive &↑↑ the risk of side effects &drug interaction
3. Proper dose
4. Antiepileptics are discontinued gradually only when the patient has been free from fits for at least 2ys

Side effects:

Allergy, GIT irritation, B.M. depression, status epilepticus, ataxia.

Blood dyscrasais →frequent Bl. Picture should be done.

- Depakin→hepatotoxic
- Epanutin → gum hyperplasia &hirsutism.

Antiepileptic drugs:

Drug group	Dose	Best indicated
1. Barbiturates: Luminal(phenobarbitone)	100-600mg\day	Broad spectrum anticonvulsant Not for petit mal
2. Hydonition: epanutin	200-600mg\d	Simple partial motor seiz. Grand mal seiz.
3. Carbamazepine: Tegritol	400-800mg\d	Simple partial motor seiz. Complex partial motor seiz. Grand mal seiz. Not for petit mal
4. Clonazepam: Rivotril	2-6mg\d	Myoclonic seiz. Grand mal seiz.
5. Valproate:' depakin	600-1500mg\d	Simple partial motor seiz. Complex partial motor seiz. Petit mal seiz. Grand mal seiz. Myoclonic seiz.
6. Succinimide: Zarontin	500-1000mg\d	Petit mal seiz.
7. New anti-epileptics : – Gabapentin(neurontin) – Vigabation(sabril) – Topiramate(topamax) – Lamotrigine(lamictal) – Tiagabine(gabitril)	 1200-2400mg\d 2000-3000mg\d 400-800mg\d 200-600mg\d 30-60mg\d	 Refractory partial &generalized seiz Refractory partial &generalized seiz Refractory partial &generalized seiz Refractory partial &generalized seiz Refractory complex partial seiz

Status epilepticus

Definition:

Recurrent attacks of generalized convulsions without regain of consciousness.

It is fetal condition as it cause exhaustion, fatigue ➔any failure

Æ:

Sudden withdrawal of antiepileptic drugs.

C/P:

Generalized convulsions esp. Grand mal epilepsy.

ttt:

1. General:
   a. Managed on **IUC**→ intubated or placrd on mec. Ventilator.
   b. Guard against →fall from the bed
      →tongue clenching {mouth gage }
   c. general :
      O2 inhalation
      monitoring of: vital signs
      P_{O2} , P_{CO2} , PH & Bicarb➔{ABG }.
      CBC, antiepileptics level, Glc, electrolytes➔venous sample
      Record ECG daily .
      Start infusion of 500 ml normal saline
   d. cerebral dehydrating agents {mannitol –lasix}
2. medical :
   1) Epanutin "phetoin": 15mg/kg infusion by a rate of 50ml/min
      C.I: cardiac arrhythmia &recent MI
   2) Diazepam 5mg I.V. to be repeated every 5 min max 20mg
   3) If no improvement → phenoparbiton 2200mg infusion very slowly
   4) In resistant cases general anesthesia may be used.

Coma

Introduction:

COSCIOUSNESS is a state of awareness of self & surrounding.

The state of consciousness depends on :

1. intact ARAS (present in brain stem , hypothalamus , thalamus)
2. intact cerebral cortex

So, small lesion of ARAS OR extensive diffuse lesion of CC ➔ Coma

Etiology:

A: Intracranial causes (with signs of lateralization)

1. Trauma : cerebral contusion
2. tumor : meningioma
3. vascular : thrombosis embolism hge ,HTN encephalopathy
4. Inflammatory : meningitis, encephalitis brain abscess
5. epilepsy

N.B

All intracranial causes ➔signs of lateralization except HTN encephalopathy

B: Extracranial causes:

1. ***Toxic:*** atropine ,aspirin ,alcohol
   - Barbiturates
   - CO poisoning
   - Morphine
2. ***Hypoxic:*** Pulmonary disease.
3. ***Ischemic :*** cardiac arrest ,MI ,hypotension
4. ***Metabolic :*** DM ,LCF ,RF ,collagen disease
5. ***endocrine :*** DM ,Hyper&Hypothyroidism ,Hyper&Hypoparathyroidism ,Addison´s crisis
6. ***Fever*** **(**febrile coma**)**

Febrile coma:

- ***Infective*** *: encephalitis , meningitis ,UTI ,pneumonia*
- ***Vascular*** *: subarachenoid hge*
- ***Toxic****: Atropine ,aspirin ,Halothane(malignant hyperthermia).*
- ***Metabolic*** *: Hyperthyroidism ,Addisonian crisis ,DKA.*
- ***Sun stroke***

Diagnosis of a case of coma:

1. **clinical exam.:** 1 Degree
   2 Etiology
2. **Investigations for the cause.**

Degree of coma

I: Old classification:

1. **Drowsiness**(lethargy) :state of disturbed consciousness in which the patient can be aroused to call
2. **Stupor: patient** can be aroused by only vigorous &repetitive painful stimulation & return to deep sleep when not continually stimulated.
3. **Semi coma :** complete loss of consciousness with a response only at the reflex level
4. **coma :** un arousable , unresponsiveness to all stimuli

II: Glasgow coma scale :

Eye opening	Verbal response	Motor response
Spontaneous…4	Oriented ……..……..5	Obey commands...6
To speech ……..3	Confused ……..4	Localize to pain ….5
To pain …….2	Inappropriate words…...3	withdraw from pain 4
None……..1	Inappropriate sounds…2	Flex to pain …….3
	None ……..…1	Extend to pain ……2
		None ……..1

Mild : 13-15

Moderate: 9-12

Sever : ≤8

Approaches to Diagnosis:

I. **History :** from relatives
- ✶ About mode of onset :
  Sudden: Hge.& Embolism

  Gradual: Thrombosis & DKA
- ✶ History of DM _ Epilepsy _ Drug intake

II. **Examination :**

☑ **Temp:**
- Hyperthermia :see febrile coma
- Hypothermia : hypothyroidism _ Morphine

☑ **Pulse:**
- ↑↑hyperthyroidism
- ↓↓hypothyroidism

☑ **BL.Pr:**
- ↑↑in HTN encephalopathy
- ↓↓in Addisonian crisis

☑ **Odour of breath**
- Acetone→ DKA
- Uriniferous→CRF

☑ **Respiration:**
- Rapid deep respiration → DKA
- Slow deep respiration.→ Morphine

☑ **Skin:**
- Dry→ DKA
- Moist→hypoglycemic coma
- Needle markers→ addiction

☑ **Pupil:**
- Dilated : atropine poisoning (bilateral)
- Constricted : morphine , metabolic coma

☑ **CNS: Signs of lateralization ☠☠**
- ***Unequal pupils***
- ***Facial asymmetry***
- ***Turning of the head to one side***
- ***Unilateral hypo. Or hypertonia***
- ***Unilateral Babinski***
- ***Unilateral jacksonian fits***
- ***Asymmetrical deep reflexes.***

Investigations:

Intra Cranial: CT, MRI, CSF…...

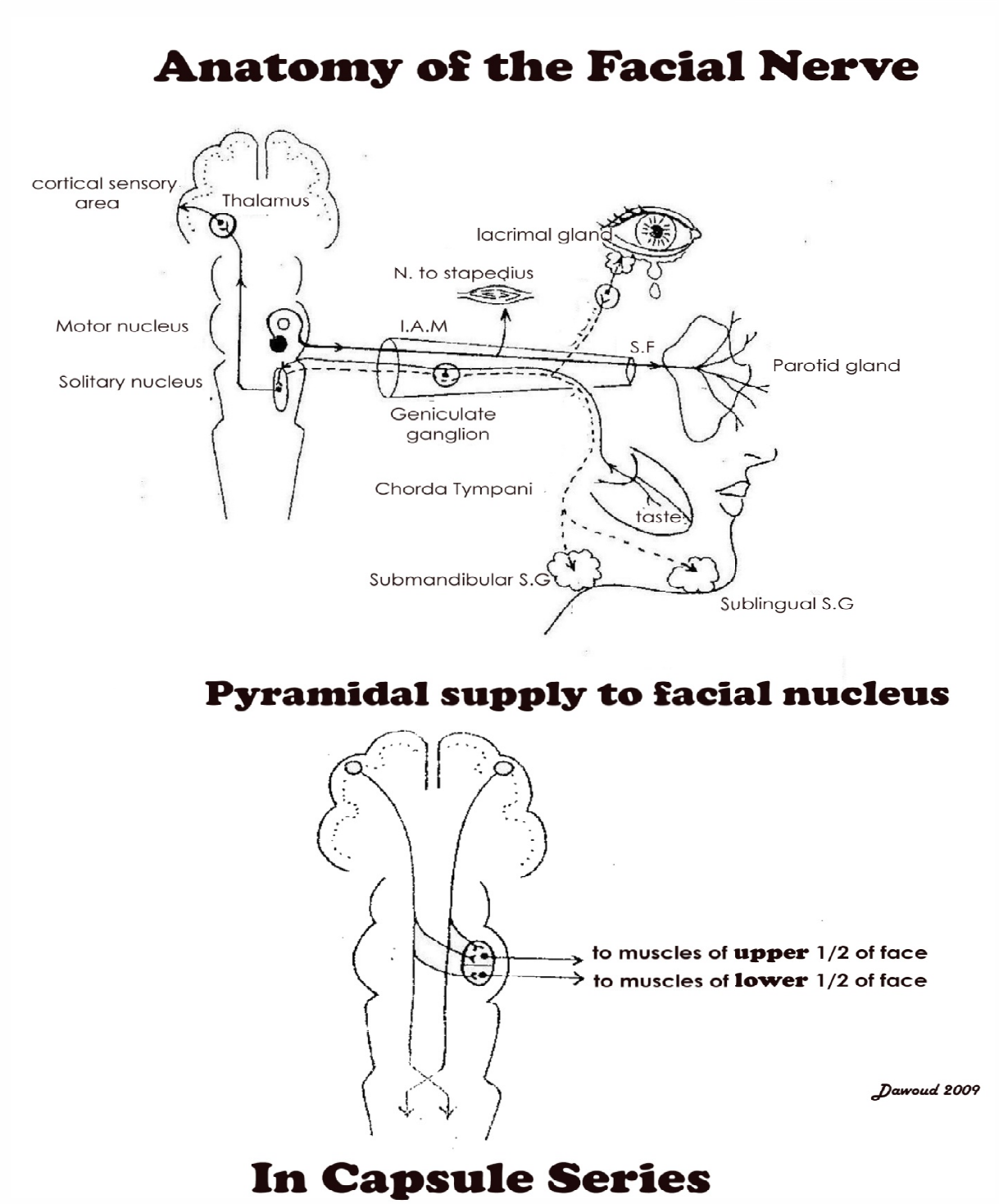
Extra Cranial : ***in basis of the most probably cause***

Treatment:

***General care*: 5B** →see hemiplegia

ttt of the cause.

Facial nerve



It is a mixed nerve as it contains:

1) Motor fibers to the following muscles:
   - ⇨ Orbicularis Oculi
   - ⇨ Orbicularis Oris
   - ⇨ Frontalis muscle
   - ⇨ Buccinator muscle
2) Sensory fibers:
   - ⇨ Taste sensation from anterior 2/3 of the tongue
3) Parasympathetic fibers:
   - ⇨ for Salivary and lacrimal glands

Clinical picture:

UMNL (affecting the pyramidal tract)

1. Obliterated nasolabial folds
2. Dropping of angle of the mouth
3. Accumulation of the food behind the cheek
4. Inability to blow the cheek
5. Inability to show the teeth ☹

LMNL (affecting the motor nerve or the facial itself)

In addition to the above;

1. Inability to close the eye→ when he tries to close his eye → eye ball rolls upward (*Bell's phenomenon*)
2. Inability to rise his eyebrows

UMNL	LMNL
Features : facts see page 1	
♦ Hemiplegia on the opposite side	♦ Crossed hemiplegia
♦ Paralysis of the muscle of the lower ½ of the face on the opposite side.	♦ Paralysis of muscles of upper and lower ½ of the face on the same side (see brain stem hemiplegia)
♦ Intact emotional movements (Supplied by extra pyramidal fibers.)	♦ Paralysis of emotional movements (pyramidal and extra pyramidal)

N.B: Facial nucleus;
Upper ½ bilaterally supplied by pyramidal tract of both sides.
Lower ½ unilaterally supplied by pyramidal tract of the opposite side.

Site of lesion in LMNL

Nuclear lesion: (in pons)
- ⇨ Paralysis of all facial muscles and intact taste and salivation.

Cerebello-pontine Angle lesion.
- ⇨ Paralysis of facial muscle and diminished taste on anterior 2/3 of the tongue, salivation & lacrimation.

Facial canal lesion:
- ⇨ Paralysis of all facial muscles
- ⇨ And according to the site of the lesion;
- ✍ If **chorda tympani** is involved →(↓↓ taste sensations from 2/3 of tongue & salivation)
- ✍ If **GSPN** is involved →(↓↓ lacrimation)

BELL's palsy ☠☠

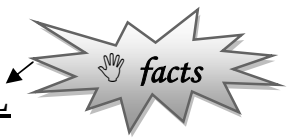
Definition:

Non-suppurative inflammation of the facial nerve just before the terminal end of facial canal →acute paralysis of the face

Etiology: Idiopathic

1. **V***ascular: Air drafts→V.C →Edema→Compression on facial nerve*
2. **V***iral: Herpes Zoster.*
3. **A***utoimmune*

Clinical picture:



Facial nerve paralysis with criteria of **LMNL**
Pain usually behind the ear
There may be, impaired sensation of taste on ant.2/3 of the tongue on the same side

***Treatment*:**

Medical: (*recovery rate 80%*)

- Cortisone (prednisolone tab. 30 mg/day)
- Vasodilators and vitamin B complex
- Protection of the cornea (eye ointment)
- Physiotherapy

Surgical: (*for the remaining 20%*)

- Decompression of facial nerve in the facial canal
- Grafting

HOW to answer

Q: Facial Nerve Paralysis??

1. Anatomy
2. Differences between UMNL & LMNL
3. Clinical picture
4. Site of lesion in LMNL
5. BELL's palsy

Studying medicine without teacher is like sailing without a boat, and studying Internal medicine without **Dr/Ahmed M.Mwafy** *is like not going to the sea at all.*

Alsayed dawoud

Kasr-Alainy school of medicine.

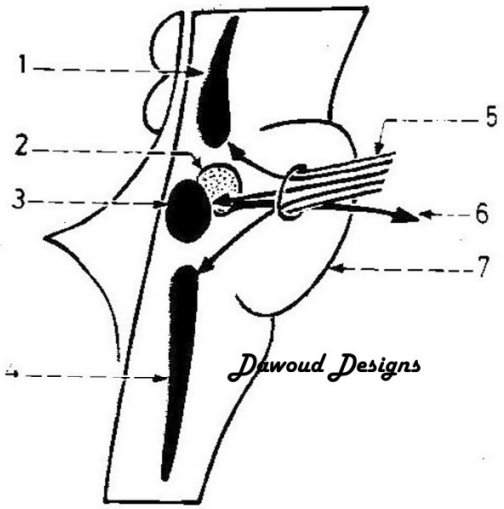
Anatomy of the Trigeminal Nerve

NUCLEI OF TRIGEMINAL NERVE

These are one motor and 3 sensory nuclei (mesencephalic, main sensory and spinal).

1. mesencephalic nucleus (in the midbrain).
2. motor nucleus (in the pons).
3. main sensory nucleus (in the pons).
4. spinal nucleus (in the medulla oblongata).
5. sensory root of trigeminal nerve (its fibres end in the 3 sensory nuclei).
6. motor root (its fibres arise from the motor nucleus).
7. pons.

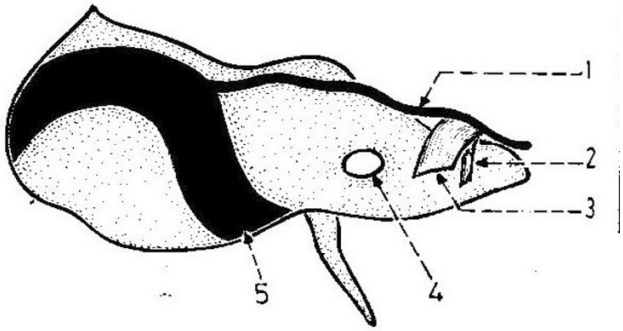
* The mesencephalic nucleus is concerned with proprioception, the main sensory nucleus with touch and pressure while the spinal nucleus is concerned with pain and temperature.



ROOTS OF TRIGEMINAL NERVE

The nerve has a large sensory root and a smaller motor root. The 2 roots cross over the upper border of the petrous bone near its apex deep to the superior petrosal sinus.

1. superior petrosal sinus.
2. motor root.
3. sensory root.
4. internal acoustic meatus.
5. sigmoid sinus.



Lesions of the Trigeminal Nerve

Sensory affection:

1) Peripheral lesions;

- Loss of sensations on the same side of the face according to the affected branch.

2) Central lesions;

\# ***Pontine lesion:***

- Weakness of the muscles of mastication
- Loss of touch sensation over the face with preservation of pain& temp. sensations.

\# ***Lesions in the medulla:***

- Loss of pain and temp. with preservation of touch e.g:
  Syringobulbia & PICA &Tabes dorsalis

N.B.

the upper part of the spinal nucleus supplies the lower face (Mandibular branch)
And the outer part supplies the inner face & Vice Versa
So, in ***Syringobulbia:*** dissociated sensory loss starting from the *outer part* of the face.
Tabes dorsalis: starting from the *central area* of the face (around the nose)

Motor affections:

- Weak muscles of mastication of **the same side**
- ***Masseter*** and ***Temporalis*** are palpated during clenching the teeth and their power can be estimated.
- ***Pterygoids:*** ask the patient to open his mouth while you fix his head.
  - Unilateral paralysis: deviation of the jaw to the diseased side
  - Bilateral paralysis: no deviation but, inability to open the mouth against resistance.

Reflex affections:

- Loss of the corneal reflex → (afferent **5** & efferent **7**)
- Loss of palatal reflex → (afferent **5** & efferent **10**)
- Exaggerated jaw reflex → (afferent **5** & efferent **5**) in bilateral **UMNL**

N.B.

Jaw reflex: when the jaw is tapped while the mouth is slightly opened nothing will appear as the reflex is too weak to be detected.

☑ Pyramidal tract lesion above the trigeminal nucleus causes the reflex to be ***exaggerated***.

Trigeminal Neuralgia (Tic douloureux)

Definition & C/P:

- Electric-shock like, lancinating pain with sudden onset, sudden termination along the distribution of one or more of the divisions of the trigeminal nerve usually 2nd & 3rd divisions
- More common in D.S, DM. and Alcoholism
- More common in ♀ ☺
- Pain is commonly evoked by shaving, smoking, talking, brushing the teeth & may also occur spontaneously

Investigations: Nothing 👎

Treatment:

Medical:

1. Tegretol 200-400 mg t.d.s
2. Phenytoin
3. Clonazepam
4. Morphine 10 mg IM. May be used

Surgical:

1. Alcohol injection into the nerve
2. Microvascular decompression

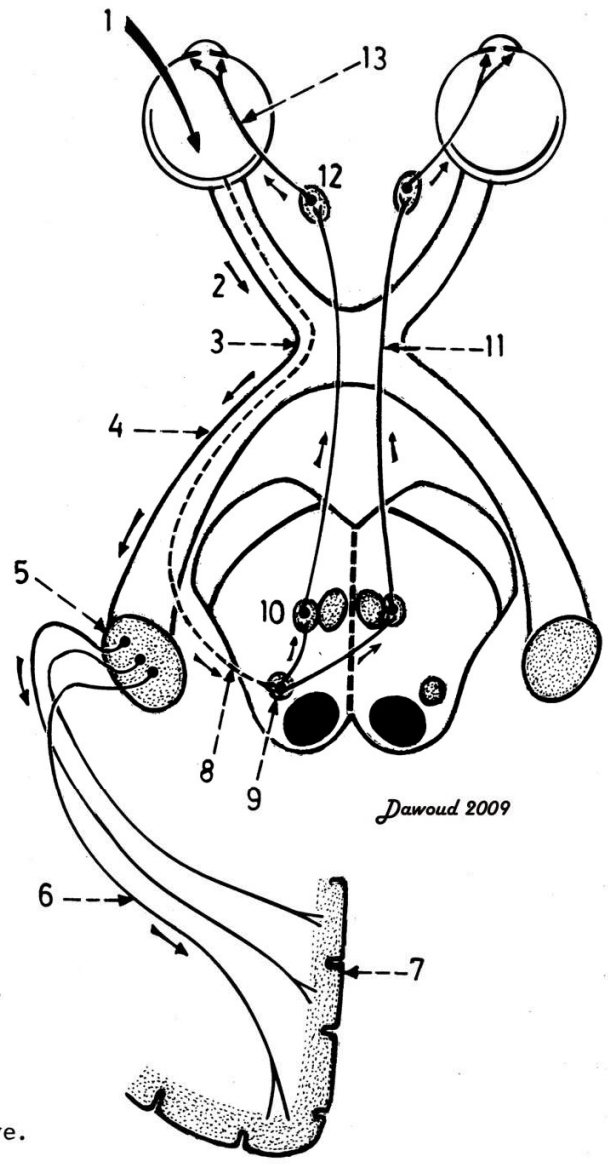
☠☠ D.D of Facial pain:

1. ***Trigeminal neuralgia.***
2. ***Diseases of teeth, sinuses, ear, nose or the throat.***
3. ***Migrainous neuralgia(Cluster headache)***
4. ***Post herpetic neuralgia***
5. ***Temporo-mandibular arthritis***
6. ***Cervical spondylosis***
7. ***Temporal arteritis***
8. ***Myocardial ischemia***

In Capsule Series

Pathway of Vesion and light reflex

The pathway of vision is as follows: retina, optic nerve, optic chiasma, optic tract, lateral geniculate body, optic radiation and occipital cortex. The pathway of light reflex is as follows: retina, optic nerve, optic tract, pretectal nucleus, Edinger-Westphal nuclei of both sides, oculomotor nerves of both sides, ciliary ganglia of both sides and postganglionic fibres to the sphincter pupillae muscle of the iris of both eyes.



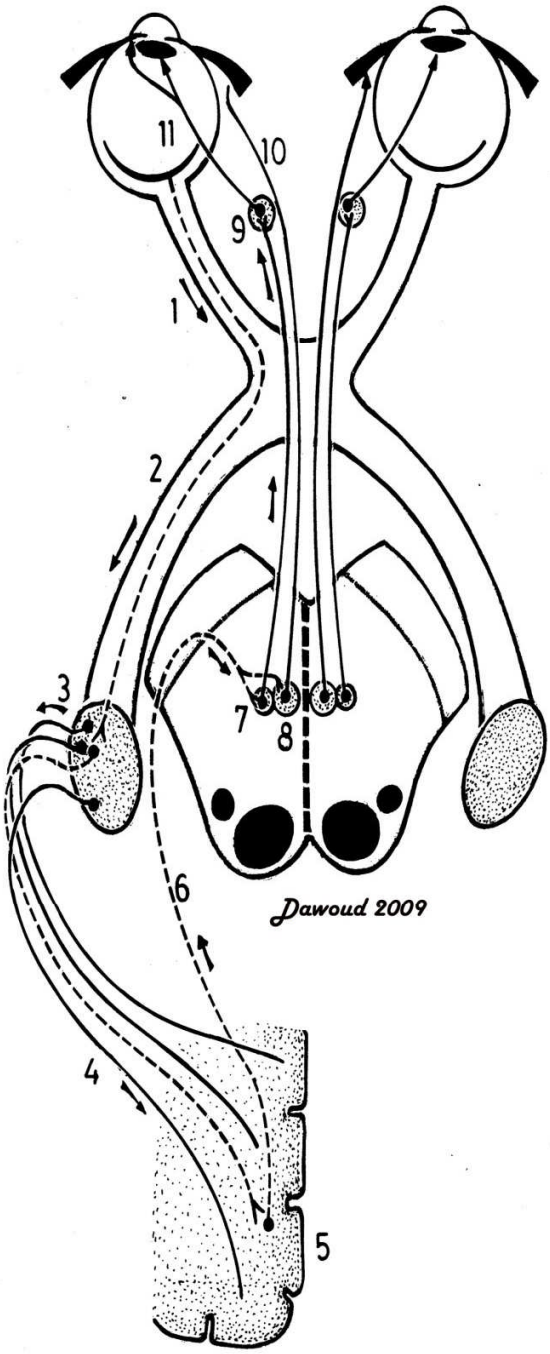
1. light falling on the retina.
2. optic nerve.
3. optic chiasma.
4. optic tract.
5. lateral geniculate body.
6. optic radiation.
7. occipital cortex.
8. fibres of light reflex leaving the optic tract.
9. pretectal nucleus.
10. Edinger-Westphal nucleus.
11. oculomotor nerve (preganglionic fibres).
12. ciliary ganglion.
13. postganglionic fibres to the sphincter pupillae muscle of iris.

* In light reflex, exposure of one eye to bright light results in contraction of the pupil of the same as well as of the opposite eye.

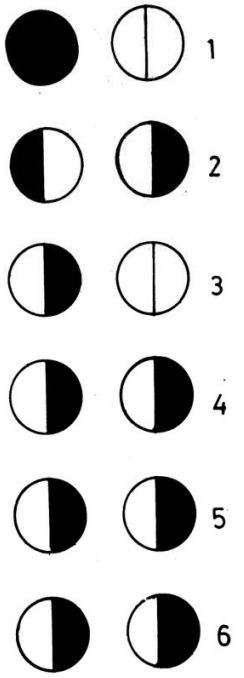
Accomodation Reflex

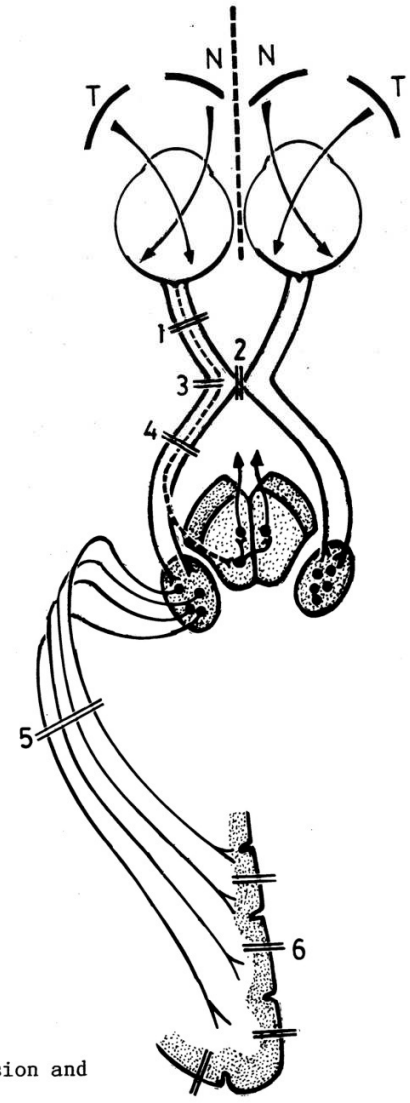
In accomodation reflex the eyes converge, the ciliary muscle contracts to modify the shape of the lens and the constrictor pupillae muscle of the iris contracts to constrict the pupil. Its pathway is as follows: retina, optic nerve, optic tract, lateral geniculate body, optic radiation and visual area in the occipital cortex. Efferent fibres from the occipital cortex pass to the oculomotor nucleus and then through the oculomotr nerve to the extra- and intra-ocular muscles.'

1. optic nerve.
2. optic tract.
3. lateral geniculate body.
4. optic radiation.
5. occipital cortex.
6. efferent fibres to the oculo-motor nucleus.
7. Edinger-Westphal nucleus.
8. oculomotor nucleus.
9. ciliary ganglion.
10. fibres of oculomotor nerve to extra-ocular muscles.
11. postganglionic fibres to intra-ocular muscles.



lesions of Visual pathway





1. lesion to optic nerve: complete loss of vision and light reflex on the same side.
2. lesion to the middle of optic chiasma: bitemporal hemianopia.
3. lesion to the lateral part of optic chiasma: ipsilateral nasal hemianopia and loss of light reflex on the same side.
4. lesion to optic tract: contralateral homonymous hemianopia and loss of light reflex.
5. lesion to optic radiation: contralateral homonymous hemianopia without loss of light reflex.
6. lesion to visual area of occipital cortex: contralateral homonymous hemianopia without loss of light reflex.

* Bitemporal hemianopia = loss of temporal fields of vision on both sides.
* contralateral homonymous hemianopia = loss of temporal field of opposite side and nasal field of same side.

Sciatica

Definition:

It is a pain along the distribution of sciatic nerve→back of thigh, leg and foot.

Causes:

1. *Acute disc prolapse (traumatic)*
2. *Lumbar Spondylosis*
3. *Malignant pelvic tumor*
4. *Sciatic nerve neuritis as in D.M.*

N.B ***the commonest cause of sciatica is disc prolapse*** **MCQ**

Clinical picture:

Symptoms:

Pain along the course of sciatic nerve ↑↑ by cough, straining, stretching

Signs:

1) **Sensory:**
   - ⇨ *hypothesia along sciatic nerve*
   - ⇨ *+ ve signs of meningeal irritation as this lead to traction on roots of sciatic nerve, pt. can't elevate the leg up to 90° without pain*
2) **Motor:** LMNL *in ms supply by the nerve*
3) **Back pain**

Investigations:

- *X- ray*
- *Myelography*
- *CT Scan*

Treatment:

1. ***Medical*** ♦ *NSAID*
   ♦ ***Ms Relaxant:*** *Norflex®* & *Glifarelax®*
2. ***Physiotherapy***
3. ***Surgery***: Decompression laminectomy

Meningitis

Definition:

- ⇨ It is an inflammation of the membrane covering the CNS
- ⇨ Inflammation of the dura is rare & is known as pachmeningitis hypertrophica $.
- ⇨ Inflammation of the pia & arachnoid is more common & known as leptomeningitis

Classification:

❶ **Acute pyogenic meningitis** *the CSF contains P.N.L*

- Meningiococci
- Haemophilis
- Streptococci
- Pneumococci

❷ **Subacute lymphocyte (non-pyogenic)** *the CSF contains lymphocytes*

- T.B.
- Viruses e.g. : herpes
- Fungi

Mode of infection:

- Blood stream from infection of other sites
- Direct from:
  - Trauma: L.P. or penetrating wound
  - Septic focus: sinusitis, otitis media, encephalitis

Meningiococcal meningitis
(acute cerebrospinal fever)

Clinical picture:

Symptoms:

- ➥ Acute onset of fever, vomiting, blurring of vision.
- ➥ Stiffness of the neck. (DD; subarachnoid hge. ***Headache*** then ***Stiffness***).

Signs:

❑ General:

- ☑ Fever 38-39 C or more.
- ☑ Rapid then slow pulse with ↑ ICT.
- ☑ Blood pressure normal except with sever cases.
- ☑ **Kgic skin** rash on the trunk due to capillary fragility (*purpura fulminans*)

Water house friedreichson's $

- ♦ *purpura due to ↑cap. fragility →purpura fulminans*
- ♦ *Adrenal gland hge → acute adrenal insufficiency & shock*

❑ Signs of meningeal irritation:

1. **Neck rigidity:** → passive flexion of the neck is difficult i.e.: *elevation of the neck and trunk together*.
2. **Neck retraction.**
3. **Brudzinski sign:**
   - ☞ Neck flexion→ flexion of both knees
   - ☞ Flexion of one hip→ flexion of the other knee
4. **Kernig's sign:**
   - ☞ pa ent in the supine posi on with his hip& knee flexed at 90
   - ☞ Try to extend the knee severe pain.
5. **Leg elevation:** →also +ve in sciatica due to affection of the root. (***Normal patient can elevate his limb up to 90 degree***).

❑ Signs of neurological deficit :

- ☑ Clouding of consciousness.
- ☑ Transient affection of the cranial nerve
- ☑ Convulsions.

Complication of meningitis :

1. **Neurological:** hydrocephalus - deaf - focal lesion as hemiplagia, aphasia with affection of the cortex (meningioencephalitis)
2. **Cardiac:** pericarditis - endocarditis.
3. **Eye:** conjunctivitis - keratitis iridocyclitis.
4. **Genitourinary:** orchitis - nephritis.
5. **Joint:** purulent arthritis.

Investigations:

- **CSF examination**
  a. ↑pressure
  b. ↓ sugar (*meningiococci consume sugar*)
  c. proteins (*Being exudate*)
  d. Purulent
  e. ↑PNL (*being pyogenic infection*)
- **Blood** → leucocytosis

Treatment:

1) ***Crystalline penicillin G:*** 24 million units/24 hrs 1/4 dose rapidly
2) ***Chloramphenicol*:** 100 mg/kg can be used in Pa ent allergic to penicillin
3) ***3rd genera on cephalosporin:*** can be used →Cefotaxime (claforan ®)
4) ***Mannitol:*** for brain edema, ***Heparin:*** for DIC.

T.B. meningitis

- Onset is insidious & preceded by Sings of toxemia
- Signs of meningeal irritation → mild.
- Usually there is a primary tuberculous focus.
- CSF→ ↑ pressure & proteins - ↓sugar - cloudy - cells are mainly lymphocytes and ↓↓ Cl markedly.
- **ttt.** Rifampicin & INH.

Encephalitis

Definition:

It is an inflammation of the brain substance.

When the spinal cord is involved it is called →Encephalomyelitis

Etiology:

Primary → *is caused by organisms which primarily attack the CNS:*

- Rabies - Polio - Inclusion body virus.

Secondary → *caused by organism which don't primarily attack the CNS but invade outside the brain then invade the nervous system:*

- **Viral:** mumps, herpes (commonest H. Simplex)
- **Parasite:** malaria.
- **Bacterial:** typhoid

Clinical picture:

❶ The onset is usually acute with ***influenza like symptoms*** including mild fever, headache

❷ ***Neurological defect:***

- ⇨ Somnolence
- ⇨ Ocular paralysis
- ⇨ extra pyramidal manifestation
- ⇨ Mental manifestations
- ⇨ Signs of meningeal irritation
- ⇨ Monoplegia or paraplegia

Treatment:

1. Proper nursing.
2. Acyclovir ® therapy for H. Simplex parental.
3. ACTH. 50 IU/d. IM→ releases cortisone.
4. Antibiotics
5. Dehydrating measures.